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Exposure and Risk Assessment for Pentachlorophenol

Little (Arthur D.), Inc., Cambridge, MA

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16. Abstract (Limit: 200 words)

This report assesses the risk of exposure to pentachlorophenol. This study is part of a program to identify the sources of and evaluate exposure to 129 priority pollutants. The analysis is based on available information from government, industry, and technical publications assembled in October 1980.

The assessment includes an identification of releases to the environment during production, use, or disposal of the substance. In addition, the fate of pentachlorophenol in the environment is considered; ambient levels to which various populations of humans and aquatic life are exposed are reported. Exposure levels are estimated and available data on toxicity are presented and interpreted. Information concerning all of these topics is combined in an assessment of the risks of exposure to pentachlorophenol for various subpopulations.

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EPA-440/4-81-021
October 1980

AN EXPOSURE AND RISK ASSESSMENT
FOR PENTACHLOROPHENOL

by

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OFFICE OF WATER REGULATIONS AND STANDARDS
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FOREWORD

Effective regulatory action for toxic chemicals requires an understanding of the human and environmental risks associated with the manufacture, use, and disposal of the chemical. Assessment of risk requires a scientific judgment about the probability of harm to the environment resulting from known or potential environmental concentrations. The risk assessment process integrates health effects data (e.g., carcinogenicity, teratogenicity) with information on exposure. The components of exposure include an evaluation of the sources of the chemical, exposure pathways, ambient levels, and an identification of exposed populations including humans and aquatic life.

This assessment was performed as part of a program to determine the environmental risks associated with current use and disposal patterns for 65 chemicals and classes of chemicals (expanded to 129 "priority pollutants") named in the 1977 Clean Water Act. It includes an assessment of risk for humans and aquatic life and is intended to serve as a technical basis for developing the most appropriate and effective strategy for mitigating these risks.

This document is a contractors' final report. It has been extensively reviewed by the individual contractors and by the EPA at several stages of completion. Each chapter of the draft was reviewed by members of the authoring contractor's senior technical staff (e.g., toxicologists, environmental scientists) who had not previously been directly involved in the work. These individuals were selected by management to be the technical peers of the chapter authors. The chapters were comprehensively checked for uniformity in quality and content by the contractor's editorial team, which also was responsible for the production of the final report. The contractor's senior project management subsequently reviewed the final report in its entirety.

At EPA a senior staff member was responsible for guiding the contractors, reviewing the manuscripts, and soliciting comments, where appropriate, from related programs within EPA (e.g., Office of Toxic Substances, Research and Development, Air Programs, Solid and Hazardous Waste, etc.). A complete draft was summarized by the assigned EPA staff member and reviewed for technical and policy implications with the Office Director (formerly the Deputy Assistant Administrator) of Water Regulations and Standards. Subsequent revisions were included in the final report.

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SECTION I.

EXECUTIVE SUMMARY

INTRODUCTION

The Monitoring and Data Support Division, Office of Water Regulations and Standards, U.S. Environmental Protection Agency is conducting an ongoing program to identify the sources of and evaluate the exposure to the 129 priority pollutants. This report assesses the exposure to and risk associated with pentachlorophenol (PCP). It is based on the collection and analysis of available data using quantitative models, whenever possible, to estimate environmental concentrations and exposure of humans and other biota, and to evaluate the overall risk. This assessment is focused on pentachlorophenol and not specifically on its contaminants and degradation products.

MATERIALS BALANCE

Production: Pentachlorophenol and its salts (e.g., sodium pentachlorophenate) are commonly used as a fungicide in wood preservatives and, to a lesser extent, as a bactericide and fungicide in cooling tower waters, rayon and textile processing, tanning, paints , glues, and in other processes or products.

Annual production of PCP is 21,300 metric tons (MT) in 1978 and occurs at only three sites in the U.S.: in the states of Michigan, Kansas and Washington. Release of the chemical to the environment during both production and transport between production sites and areas of use is apparently small, although not all transport practices have been examined. Most of the PCP produced is consumed domestically; however a small amount of PCP is exported.

Wood Preserving: Use of PCP is widely distributed throughout the U.S. Since 80% to 90% of the amount produced is used in wood preserving processes, a major portion of PCP mass is concentrated in the Southeast quadrant of the U.S., in the Mid-Atlantic states and in the Northwest where the lumber industry is located. Two principal wood preserving practices use pentachlorophenol--Boulton conditioning and steam conditioning--each associated with different efficiency in treating waste water. Only one wood preserving plant discharges PCP directly to water; others discharge to POTW's or remove the PCP in sludge during waste water treatment. Approximately 14 MT of PCP sludge are generated annually by indirect dischargers in the wood-preserving industry. The remaining plants, with no discharge, use evaporative technology and generate an estimated 60 MT of PCP in solid waste. The treated wood is widely distributed throughout the U.S. and its total contribution of PCP release to the environment is not known. As much as 344 MT may be released to air through volatilization from wood, based on estimates.

Other Uses: Other applications of PCP and its salt (that account for the remaining 10-15% of PCP use) include application to cooling tower water as a bactericide/fungicide (estimated to be 230 MT in 1978), use as a preservative in paints (approximately 800 MT of NaPCP annually) in tanning (approximately 15 MT annually), in textile processing (approximately 20 MT of NaPCP annually), and as a domestic herbicide (approximately 600 MT annually). Other uses are as a fungicide in mushroom cultivation, as a disinfectant, and as a general preservative in numerous construction materials, in the glue of food packaging, in petroleum industry products, and in photographic material. Additionally PCP has been detected in the paint of imported toys and may be present in toys manufactured overseas for domestic toy companies

Release of PCP to the Environment: The majority of PCP releases during production and use are to air (~620 MT) from wood preservers and cooling towers and to land (890 MT) from wood preservers and domestic use as a preserver and herbicide. Discharges to water, direct and through POTW's, are expected to be small: 12 MT and 5 MT, respectively. These estimates are based on available information; new data in certain important areas are likely to alter the distribution. For example, based on preliminary estimates, as much as 380 MT of PCP may be discharged into water in POTW effluents; however, this number is associated with a wide margin of error.

DISTRIBUTION OF PENTACHLOROPHENOL IN THE ENVIRONMENT

The analysis of the distribution of PCP in the environment is comprised of four parts: (1) monitoring data for PCP in the environmental media, (2) physico-chemical processes determining PCP concentrations in each environmental compartment, (3) important biological pathways and (4) estimations of PCP concentrations in air and water.

Monitoring Data: Data from monitoring of pentachlorophenol levels in various environmental media are too limited to indicate typical U.S. ambient concentrations. The published data suggest concentrations of 0.01 µg/l to 147 µg/l in surface water, both fresh and estuarine, and in some cases in industrial areas. The highest water concentrations reported were 82 to 10,500 µg/l in a culvert near a wood-preserving factory. Rainwater concentrations (in Hawaii) ranged from 0.002 µg/l to 0.284 µg/l. Sediment and organic matter in general exhibited concentrations one to three orders of magnitude higher than those in surface water. Data were not available concerning ambient PCP levels in soil and air in the U.S.

Data concerning PCP levels in urine and tissue for the general human population indicate continual exposure to pentachlorophenol. PCP has been detected in urine samples for 85% of the U.S. population. PCP residues have been reported in various food items and in fish and wildlife species at concentrations of 0-50 mg/kg.

Physico-Chemical Pathways: The air compartment appears to receive a large amount of PCP discharge as indicated by materials balance information. Pathways for entry into the atmosphere include evaporation from aeration/evaporation ponds at wood treatment facilities, evaporation and/or droplet release from cooling towers using PCP biocide, volatilization from treated wood products, and burning of treated wood. Physical removal mechanisms, such as rainout, are important processes affecting PCP concentrations in the atmosphere. Photolysis also appears to be an important transformation process on the basis of PCP's behavior in water. The atmospheric lifetime for PCP is estimated to be one month and is assumed to be determined primarily by physical removal processes.

Releases to surface water occur through direct discharge and direct entry from numerous non-point sources including treated wood. In addition, PCP can be transferred to water from other compartments such as air through rainout, and soil, throughout runoff and leaching. At low concentrations in water, PCP appears to be quite readily degraded by microbial action (in water treatment facilities) and, in clear water, photolyzes rapidly. Extreme concentrations or turbid conditions could inhibit these two processes. Sorption onto sediments and larger organic matter is also likely to occur. Based upon laboratory studies, hydrolysis, oxidation and volatilization do not appear to affect concentrations in water significantly. The lifetime of PCP in natural waters is estimated to be one week in conditions that are optimal for photolysis. For systems in which biodegradation is the primary removal mechanism, the lifetime will be longer and extremely variable, depending on environmental conditions. Once PCP is adsorbed onto sediment, there appears to be potential for re-release to water; however, the conditions that would trigger this transfer have not been investigated.

Pentachlorophenol enters the soil compartment as a result of its use as a garden herbicide, leaching from treated wood, rainout from the atmosphere, and spills around PCP-using industries. Due to its high adsorptivity, especially on organic matter and under acidic conditions, and based on the results of one laboratory study, PCP does not appear to be very mobile in soil. Biodegradation is a significant removal process under both aerobic and anaerobic conditions. Important variables affecting the degradation rate include temperature, moisture, the presence of organic matter, and to a lesser degree, the soil cation exchange capacity and pH. Photolysis and volatilization do not appear to be important determinants of PCP concentrations in soil.

Wood that has been treated with pentachlorophenol apparently loses, at a minimum, the amount of chemical residing in the wood's surface layer. Photolysis may degrade PCP in products exposed to sunlight, while volatilization occurs at a fairly slow rate, estimated at $1.7 \times 10^{-10} g/cm^2/sec$. On the basis of this volatilization rate, a lifetime of one year for PCP residence in wood is estimated. The potential exists for removal of PCP from treated wood by runoff from surfaces or leaching, however, the overall importance of this process could not be assessed.

Biological Pathways: PCP is accumulated in the tissues of biota at concentrations greater than water concentrations by a factor of one to 1000. Depending on the species, some of the PCP is metabolized and may be concentrated in specific organs, such as the liver. Certain plant species take up PCP and, some species rapidly metabolize the compound.

Calculated Concentration Estimates: Using simple quantitative models, estimates were made of ambient air concentrations resulting from the following sources: wood treatment waste water evaporation ponds, volatilization from treated wood, evaporation from cooling towers, and open-burning of treated wood. Estimated levels were 4.3 ng/m^3, 56.0 ng/m^3, 36.0 ng/m^3 and 40.0 ng/m^3, respectively, summing to 136.3 ng/m^3. Localized concentrations in the immediate vicinity of a cooling tower plume and a waste water evaporation pond were estimated to be approximately 10^4 ng/m^3, and 10 ng/m^3, respectively.

With use of the computerized EXAMS model, estimates were made of surface water concentrations generated by three discharge rates--0.7, 0.25 and 17.1 kg PCP/day--into six generalized aquatic ecosystems. The calculated water column (total) concentrations ranged from 0.003 µg/l for the lowest pollutant loading into a river system to a high of 26,000 µg/l for the highest pollutant loading into a pond system.

HUMAN EXPOSURE TO AND EFFECTS OF PCP

Exposure: Incidences of PCP exposure and toxicity to humans have been reported for both occupational and non-occupational populations. Most exposure is occupational involving dermal contact or inhalation of contaminated water vapor. Eighty-five percent of the general U.S. population has PCP residues in the urine indicating continual exposure to the chemical. Potential routes of exposure include ingestion of food or drinking water, inhalation and dermal absorption.

Ingestion is associated with relatively low per capita exposure levels: in food 0.76-1.5 mg/day on average and 18 mg/day maximum; in drinking water 0.02 µg/day average and 24 µg/day, maximum. Certain foods with high PCP concentrations include fish, with a maximum exposure level of 0.1 mg/day, peanut butter with 6.2 mg/day and gelatin with 0.02 mg/day (high PCP level based on limited sample). The subpopulation exposed to the maximum levels is expected to be small.

There are numerous potential exposure routes through inhalation of PCP-contaminated air--both occupational and non-occupational. Exposure levels of 0.9 mg/day to 14.4 mg/day were estimated for workers in wood treatment plants. In the vicinity of a cooling tower (<1 km), workers may be exposed to 14.4 mg PCP/day. Domestic use of PCP in spray form results in an estimated exposure level of 0.91 mg/day.

The general population is expected to be exposed to ambient levels of PCP in the air resulting from wood treatment waste water evaporation (0.09 µg/day), volatilization from treated wood (1.1 µg/day), evaporation from cooling towers (0.7 µg/day), and open-burning of treated wood (0.8 µg/day). The total exposure level from all sources combined is 2.7 µg/day.

Certain subpopulations may be exposed to higher concentrations of PCP downwind from cooling towers (maximum 2.0 mg/day) and from waste water evaporation ponds (0.002 mg/day). Subpopulations using PCP indoors on wood panelling may be exposed to 3.2 mg/day through inhalation of volatilized PCP.

Exposure to PCP may also occur through dermal absorption. Contact with a contaminated water supply has an associated maximum exposure level of 0.48 µg/day. Direct spilling of PCP on the skin may result in an exposure level of 170 mg per incident and handling of treated wood, 0.5 mg/day.

Effects: Determination of the effects of pentachlorophenol is complicated by its contamination with varying amounts of polychlorinated dibenzo-furans. These contaminants and other impurities may comprise from 1% to 15% of the total PCP. However, purified PCP appears to be toxic by itself as indicated by fetotoxicity and teratogenicity studies.

Teratogenicity and fetotoxicity are the most serious effects of PCP indicated by available data. Adverse effects were observed following exposure of rats to 30 mg/kg day during gestation. Purified PCP was more toxic than technical grade PCP with respect to certain of the observed effects. A no-effects level of 6 mg/kg/day has been observed; OPTS reports a lower level of 3 mg/kg/day for fetotoxic effects. Carcinogenicity and mutagenicity data are much less conclusive due to the small number of experiments and conflicting data. Chronic dietary exposure to 100 ppm of technical grade PCP has caused liver lesions in rats. The median oral lethal dose for rats is 78-205 mg/kg.

Two other routes of exposure, inhalation and dermal absorption, are important. Inhalation of PCP aerosol has an associated LD_{50} of 11.7 mg/kg in rats. Dermal LD_{50}'s reported for rats range from 96 mg/kg to 320 mg/kg.

NON-HUMAN EXPOSURE TO AND EFFECTS OF PCP

Exposure: Aquatic organisms are typically exposed to PCP in water at concentrations less than 100 µg/l. Simulations of water concentrations resulting from practiced discharge rates are in general agreement with these exposure levels, with the exception of discharges to the pond system and at the highest loading rate to the eutrophic lake system. Higher water concentrations have been measured in industrial areas, the highest at 10,500 µg/l. Even higher concentrations may be found associated with oil slicks on water surfaces near sources. Incidences of fish exposure to PCP have been reported in the vicinity of wood preserving plants, pulp and paper industries, cooling water discharges, and following spraying.

Terrestrial organisms may be exposed to PCP through contact with spilled material and ingestion of contaminated plants and aquatic organisms. No information regarding actual exposure was available.

Effects: The lowest reported effects level for freshwater aquatic organisms was 1 μg/l in algae. Sock-eyed salmon exhibited sublethal effects at 1.74 μg/l. The lowest acute effects level was an LC_{50} of 15.5 μg/l for rainbow trout. LC_{50}'s reported for freshwater fish and invertebrate species ranged over two orde~~rs~~ of magnitude.

Marine aquatic organisms exhibited lowest effects, at 38 and 43 μg/l for a fish and invertebrate species, respectively. Only limited toxicity data were available concerning both acute and chronic effects.

Several water parameters influence PCP toxicity. Toxicity increases with decreasing pH, increasing temperature and, to a lesser degree, decreasing hardness.

No information was available concerning the effects of PCP on terrestrial animals and only limited data on phytotoxicity were found.

RISK CONSIDERATIONS

Risk to Humans: The non-occupational human population potentially at risk with respect to PCP can be divided into three categories: 1) the general U.S. population, 2) subpopulations residing in the vicinity of PCP sources, and 3) subpopulations directly using PCP as a preservative, bactericide or herbicide. Calculating margins of safety--the ratio of the exposure level to the lowest effect level (in this case, of a no-effects level for rats of 3 to 6 mg/kg)--gives an indication of the significance of the exposure level in relation to toxicity. The general population is potentially exposued to PCP through ingestion of food (0.3 mg/kg/day maximum) and drinking water (0.0004 mg/kg/day maximum), dermal absorption of water (negligible), and inhalation of nationally distributed air concentrations (0.00005 mg/kg/day). All of these routes are associated with low exposure levels compared to the following levels. Subpopulations residing near PCP sources such as cooling towers and wood preserving waste water evaporation ponds, are exposed through inhalation to higher levels estimated at 0.001 mg/kg/day to 0.03 mg/kg/day depending on proximity to source. Subpopulations using PCP directly are exposed to levels estimated at 0.03 mg/kg/day from use of spray, 0.05 mg/kg/day from breathing fumes from treated wood indoors, and 2.8 mg/kg from a direct spill on the skin.

Combining several exposure routes--ingestion, inhalation and absorption--gives a maximum exposure level of 3.2 mg/kg/day if a direct spill on skin is included; this exceeds the no-effects level set by OPTS. Eliminating the spill gives a total exposure level of 0.4 mg/kg/day. A margin of safety of 8.6 is associated with this combined exposure level. Most other individual exposure routes have associated margins of safety of at least 100; however, due to the uncertainties of such an assessment, these are not meant to indicate acceptable levels of exposure.

In addition to the described exposure routes, there is limited epidemiological evidence possibly linking PCP exposure through the handling of treated packing cases with a higher incidence of leukemia in Kentucky. The results of further investigation of this possible relationship are expected to be published in the near future.

Risk to Other Biota: Aquatic organisms are likely to encounter surface water PCP concentrations that may have acute adverse effects on a few species (certain algae and salmonids) and sublethal effects on a larger number of species. Acute effects were reported for concentrations ranging from 15.4 µg/l to over 100 µg/l and many ambient water concentrations reported also fell within this range. Incidents of fish kills are common, but usually occur in the vicinity of obvious PCP sources, probably resulting from concentrations much higher than 100 µg/l. The paucity of monitoring data makes assessment of risk to aquatic organisms difficult because of lack of information on regional distribution of ambient environmental concentrations of PCP.

The limited monitoring data base indicates ambient surface water concentrations ranging from 0.001 µg/l to 100 µg/l, with numerous observations greater than 1.0 µg/l especially in the Pacific Northwest. No data are available for many of the U.S. major river basins, such as in the Southeastern and South Central states, where PCP concentrations are expected to be high.

Fish kill data and miscellaneous observations of high PCP concentrations in water implicate several industries and activities that may generate levels of PCP potentially harmful to aquatic life. These include the wood preserving industry (through holding lagoon spills, runoff from contaminated soil), the pulp and paper industry, cooling water discharges and spraying of wood near water. The results of the analysis with the EXAMS model indicate potentially harmful water concentrations generated by continuous discharge of 0.25 kg/day into static aquatic systems.

Certain PCP sources warrant further investigation, such as leaching from treated wood products and PCP-treated paint on products in contact with water. In addition, information is needed on typical PCP concentrations in effluents from and the discharge practices of PCP-associated industries.

SECTION II.

INTRODUCTION

The Office of Water Regulations and Standards, Monitoring and Data Support Division, of the U.S. Environmental Protection Agency is conducting a program to evaluate the exposure to and risk of 129 priority pollutants in the nation's environment. The risks to be evaluated include potential harm to human beings and deleterious effects on fish and other biota. The goal of the task under which this report has been prepared is to integrate information on cultural and environmental flows of specific priority pollutants and estimate the risk of receptor exposure to these substances. The results are intended to serve as a basis for evaluating the magnitude of the risk and developing suitable regulatory strategy for reducing any such risk when action is warranted.

This report provides a brief, but comprehensive, summary of the manufacture, use, distribution, fate, effects and potential risk of pentachlorophenol (PCP). Making effective use of the information given requires an understanding of uncertainties regarding the data and qualifications regarding conclusions presented herein.

Pentachlorophenol has numerous uses. The majority of the chemical produced is used in wood preserving, and a significant amount of data has been collected concerning this use. However, other uses must be considered also because their nature provides a greater potential for exposure to receptors. Very little information is available for certain of these alternative, yet significant applications regarding the total quantity involved and typical use concentrations. This lack of information makes comparison between different exposure pathways uncertain (e.g., exposure to PCP in cooling towers vs. treated wood).

Although pentachlorophenol is widely used throughout the U.S., very little monitoring data exists for levels in soil, water and air. In order to provide the information necessary for exposure analysis, estimations of concentrations in water and air were calculated using materials balance as input. The calculations are based on simple calculational models and many assumptions. Since persistence of PCP in any environmental medium is a complex phenomenon determined by many site-specific and largely non-generalizeable parameters, the calculated values may not be indicative of actual concentrations in specific environmental systems.

Pentachlorophenol has associated with it a number of impurities, including dibenzofurans and chlorinated dioxins. The degree of contamination varies by the PCP product formulation or grade, by the company producing it, and even by batch of chemical. Although little information is available describing the toxicity of these compounds. related compounds are known to be highly toxic (e.g., TCDD). Most toxicology investigations concern the effects of one of the commercial formulations of PCP and do not distinguish between toxicity due to PCP itself and that due to its contaminants. Some of the variability in reported toxic concentration

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thresholds can be attributed to variability in levels and types of contaminants in the material tested. The contaminants in PCP may be important determinants of the observed effects of exposure to various grades of PCP. However, the level of effort involved and the limitations of currently available data precluded distinguishing the risks of exposure to PCP from the risks of exposure to the accompanying contaminants. Therefore, the presence of contaminants in PCP introduces another element of uncertainty into the analysis.

The report is organized as follows:

Section III presents a materials balance for PCP that considers quantities of the chemical consumed in various applications, the form and amount of pollutant released to the environment, the environmental compartment intially receiving it, and, to the degree possible, the locations and timing of releases.

Section IV describes the distribution of PCP in the environment by presenting available monitoring data for various media, by considering the physicochemical and biological fate processes that transform or transport PCP, by calculating concentrations for several critical environmental pathways.

Section V describes the available data concerning the toxicity of PCP for humans and laboratory animals and quantifies the likely level of human exposure via major known pathways.

Section VI considers toxicological effects and exposure to non-human biota, predominantly aquatic biota.

Section VII presents a range of exposure scenarios for humans and likely exposure conditions for other biota and correlates these with reported effects levels from Sections V and VI, in order to assess the risk presented by various exposures to PCP.

Appendix A discusses the contaminants found in different grades of pentachlorophenol.

Appendix B presents an overview of available information concerning PCP biodegradation pathways and products.

SECTION III.

MATERIALS BALANCE

A. INTRODUCTION AND METHODOLOGY

In this section, a materials balance is developed for pentachlorophenol. The materials balance considers pentachlorophenol as it is released from the cultural environment to its first point of entry into the natural environment. Potential sources of pentachlorophenol releases were identified by a review of activities in which the material participates from its production and use in various forms to its ultimate disposal.

For each major source of pollutant release, the amount of material released was estimated, and the environmental compartments (air, land, and water) initially receiving and transporting the material were identified, as were the locations at which the pollutant loadings take place. There are many uncertainties inherent in this type of analysis: not all current releases have been identified, past releases were not well documented, and future releases are difficult to predict. Nevertheless, sufficient information is available to indicate in general terms the nature, magnitide, location, and time dependence of pollutant loading of the environment with pentachlorophenol.

B. MATERIALS BALANCE

Pentachlorophenol is produced in the U.S. by chlorination of phenol in the presence of a catalyst; it can also be formed by the hydrolysis of hexachlorobenzene, but this process is currently not used. In addition to several trade names, the material is commonly referred to as "penta" and PCP.

Pentachlorophenol is toxic to a wide spectrum of life forms, including bacteria, algae, fungi, and certain plants. Because of this property, over 80% of the pentachlorophenol and its co-product, sodium pentachlorophenate, produced is consumed in the timber and plywood industry as a wood preservative. PCP is also applied as an industrial and commercial pesticide and acts as a preservative in paints for wooden items such as lawn furniture, trailers, and boats.

In 1978, total U.S. industrial demand was 21,300 metric tons (MT) (U.S. International Trade Commission, 1979). Table 1 shows U.S. production and consumption patterns for that year. Estimates of environmental releases resulting from production and consumption of PCP are presented in Table 2, and Figure 1. Manufacture of pentachlorophenol is expected to increase by 2-3% each year through 1981. The volume of future use of the chemical is somewhat uncertain due to increasing concern about its potential for adverse environmental effects.

TABLE 1. SUMMARY OF U.S. PRODUCTION AND CONSUMPTION OF PENTACHLOROPHENOL, 1978

Source/Consumer	Supply (MT) Year	Consumption (MT)
Production--1978	18,140	
Domestic Stocks--1978	3,160	
Wood Preserving--1978		18,000[2]
Sodium-PCP		2,500[3]
Cooling Towers		(230)[4]
Paints		(800)
Textiles		(20)[4]
Tanning		(15)[4]
Home and Garden		600
Herbicide		200
Total	21,300	21,300

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[1] United States International Trade Commission, 1979
[2] Linsey, 1979
[3] Versar, 1978
[4] MITRE, 1979

( ) Indicates most of this quantity is sodium PCP, therefore, should not be included in PCP consumption total.

TABLE 2. SUMMARY OF ENVIRONMENTAL RELEASES OF PENTACHLOROPHENOL (ESTIMATED 1978)

Source	Release (MT/Yr)			
	Air	Direct Aquatic	POTW	Land
Production	50	*	*	*
Wood Preserving	-	insignf[2]	5.3[1,2,3]	74.5[1,2,3]
Preserved Wood	344	**	-	*
Production of NaPCP	*	*	*	*
Cooling Towers	228.0	2.0	†	-
Paints	-	-	-	-
Textiles/Rayons	-	3.0	†	9.0
Pulp and Paper Mills	-	5.0	-	-
Tanning	-	2.0	†	6.0
Home and Garden	**	**	**	600.0
Herbicide	**	**	**	200.0
POTW	-	-	-	18.0
Total	622	14.5	5.3	881.1+

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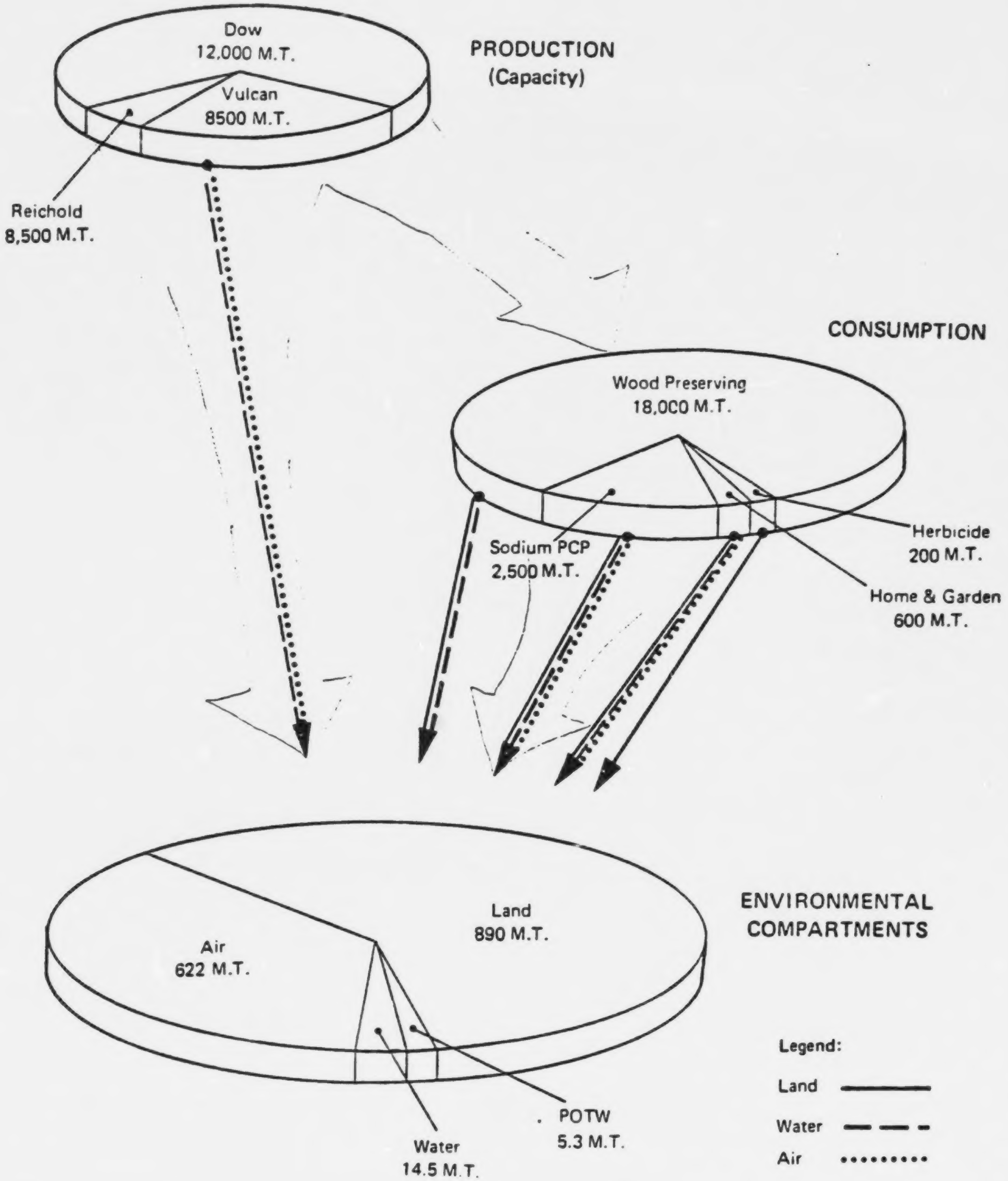
[1] Arthur D. Little, Inc., industrial experts.
[2] EPA, October 1979.
[3] Rao, 1978.

[*] At this time, not enough sampling has been done to permit estimates. of gross environmental discharge.

[**] Portions of these releases enter directly or indirectly through the water compartment through groundwater, surface runoff, etc.; however, due to the uncertain nature of the release, the contribution to water cannot be quantified.

[†] It is uncertain whether these industries discharge directly to water or through POTW's.


Dow
12,000 M.T.
Vulcan
8500 M.T.
Reichold
8,500 M.T.
PRODUCTION
(Capacity)
CONSUMPTION
Wood Preserving
18,000 M.T.
Sodium PCP
2,500 M.T.
Herbicide
200 M.T.
Home & Garden
600 M.T.
ENVIRONMENTAL
COMPARTMENTS
Land
890 M.T.
Air
622 M.T.
Water
14.5 M.T.
POTW
5.3 M.T.
Legend:
Land
Water
Air

Note: Boundaries between receiving medium are often undefined and/or changing; PCP apparently released to one compartment can result in another

Source: Arthur D. Little, Inc., based on 1978 estimates

FIGURE 1 MATERIALS BALANCE OF PENTACHLOROPHENOL

1. Production

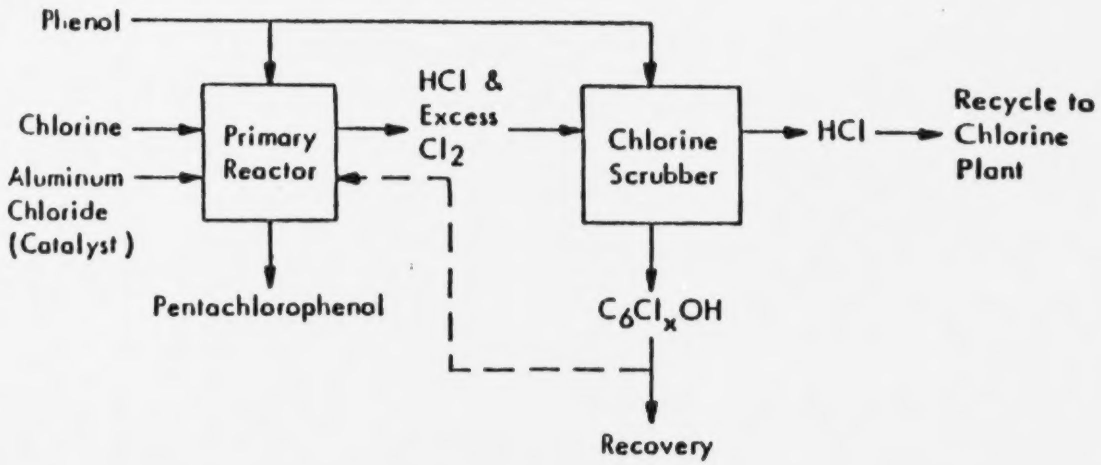
The majority of pentachlorophenol consumed in the United States is produced domestically and only a small portion of U.S. production is exported. The chemical is produced industrially in a two-stage process, shown schematically in Figure 2. PCP is formed by chlorinating phenol at close to atmospheric pressure. In the first stage of production in the primary reactor, phenol is maintained at a temperature between 65°C and 130°C until the solution stabilizes at 95°C. At this stage, three to four chlorine atoms are joined while the reactor temperature is held about 10°C above the final product melting point. The entire process is completed in 5-15 hr.

A byproduct of the chlorination product is an off-gas (HCl initially and chlorine gas in the later stages), which is treated by a scrubber-reactor system utilizing excess phenol. Some of the lower chlorinated phenols formed are recovered and returned to the primary reactor and the remaining vapor is pure HCl gas.

Until 1978, there were four pentachloraphenol producers in the United States. Dow Chemical, U.S.A., has an estimated annual capacity of 12,000 MT in Midland, Michigan, using equipment exclusively designated for PCP production; Dow recycles HCl in the process. Reichhold Chemicals, Inc., has the estimated capacity to produce about 8,500 MT of PCP annually at a facility located in Tacoma, Washington. Vulcan Materials Company, Chemicals Division, has a capacity of about 8,500 MT in Wichita, Kansas. Monsanto Company's Industrial Chemical Company had a plant capacity of 12,000 MT in Sauget, Illinois, manufacturing phenol in its Gulf Coast plant; however, Monsanto stopped producing PCP in 1978 due to a shift in market demand and in order to streamline operations (Linsey, 1979).

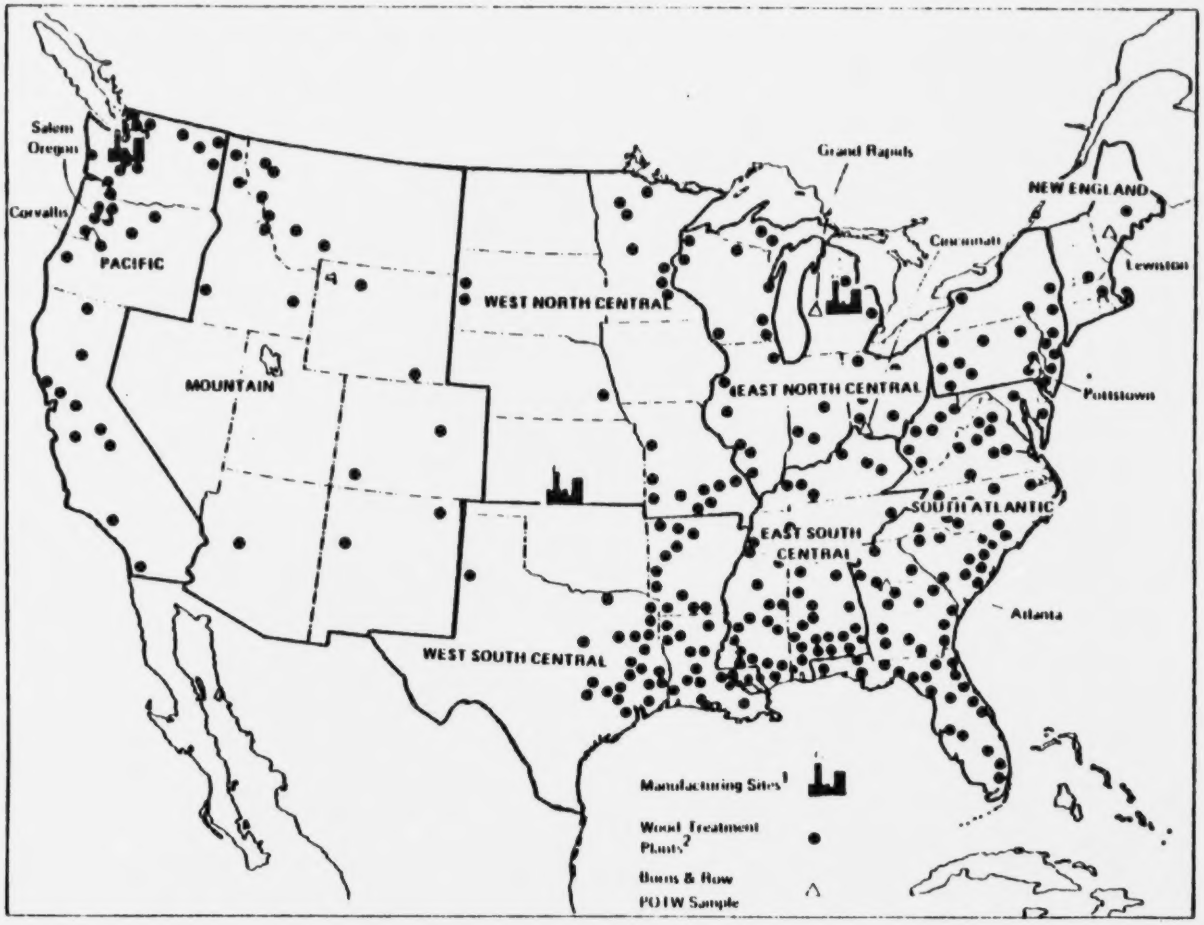
Pentachlorophenol production has been steady at approximately 21,500 MT (Linsey, 1979) despite a brief decline in production following a contamination incident in Michigan involving cattle exposed to PCP-treated wood in their shelters. Following Monsanto's shutdown, compensating increases in production by the other three companies have maintained a constant production volume.

Emissions during production are relatively insignificant in volume and are geographically restricted to the three currently operating facilities shown in Figure 3 (Versar, 1979). As described, air emissions are limited by a scrubber mechanism enabling recovery. Incentives for control are economic, as well as regulatory. An additional release potential during production is discharge of contaminated cooling waters. These are treated prior to release; however, industries producing and consuming PCP have not yet been subjected to effluent guidelines and, consequently, no sampling data are available upon which to base estimates of gross annual discharge.


Phenol
Chlorine
Aluminum Chloride (Catalyst)
Primary Reactor
Pentachlorophenol
HCl & Excess Cl_2
Chlorine Scrubber
HCl
Recycle to Chlorine Plant
C_6Cl_xOH
Recovery

Source: U.S. EPA 1974.

FIGURE 2 PRODUCTION OF PENTACHLOROPHENOL


Salem Oregon
Corvallis
PACIFIC
MOUNTAIN
WEST NORTH CENTRAL
Grand Rapids
EAST NORTH CENTRAL
Cincinnati
NEW ENGLAND
Lewiston
Pottstown
SOUTH ATLANTIC
EAST SOUTH CENTRAL
Atlanta
WEST SOUTH CENTRAL
Manufacturing Sites[1]
Wood Treatment Plants[2]
Burns & Roe POTW Sample

Notes: 1. Battelle for EPA, 1975
2. Forest Service, U.S. Department of Agriculture

FIGURE 3 LOCATIONS OF PULP MANUFACTURING PLANTS AND WOOD TREATMENT PLANTS

Normally PCP is shipped in two forms: first in reusable containers or drums, which are returned to the manufacturer following delivery and to be recycled exclusively for PCP transport; and second, in a large cake weighing around 2 MT, which is handled by mechanized equipment and transported in trucks.

Previous studies of pentachlorophenol use and emissions have suggested that material may be released during or as a result of transport from the manufacturer to the consumer. Aside from identifying this possible source of release, however, prior reports have been unable to quantify the release to the environment and have suggested exploring methods of transport and, in particular, procedures used by the railroad companies to clean freight cars after use.

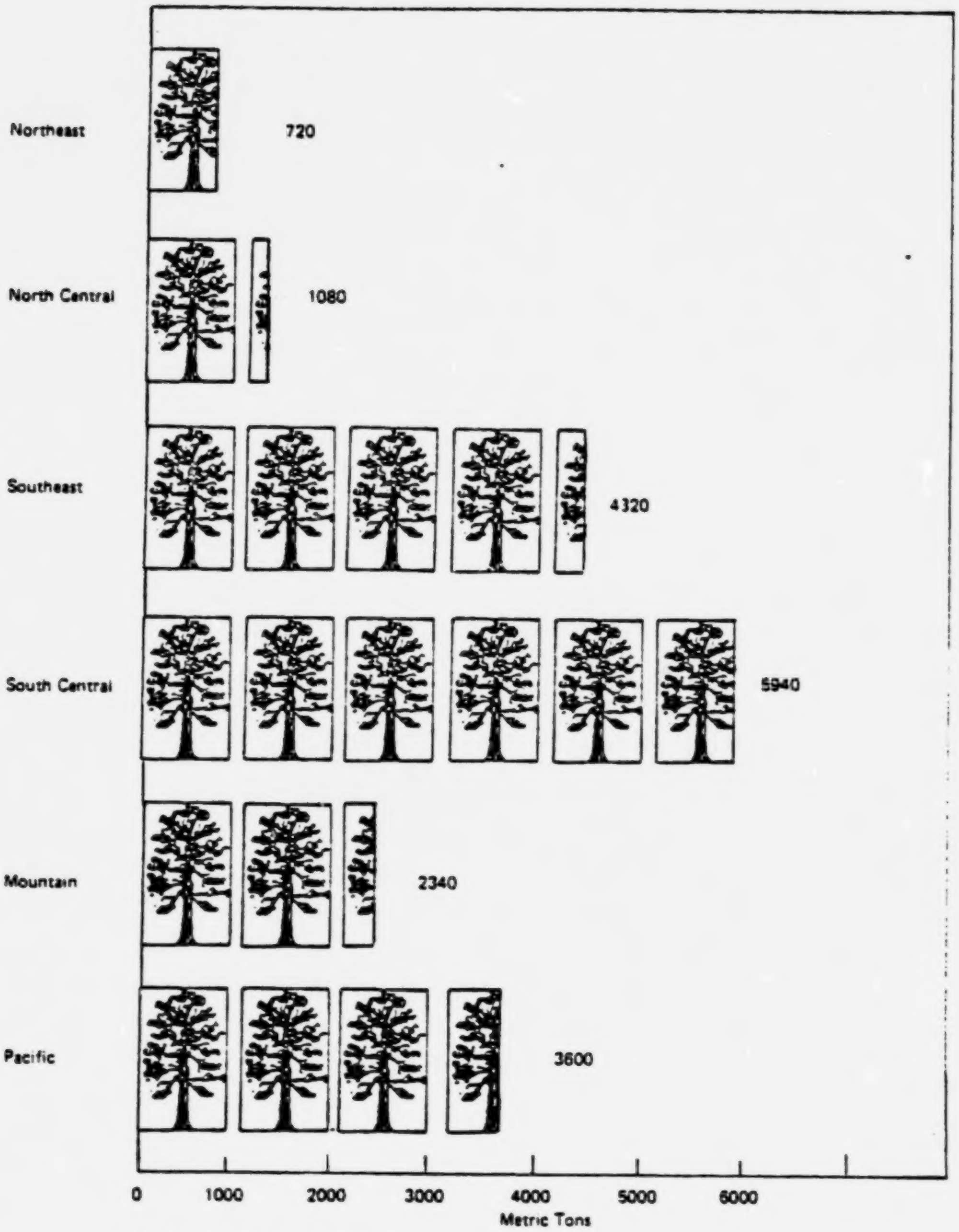
Since PCP is not currently shipped in raw form in railroad freight cars, this type of release is no longer occurring. This has been verified by the Forest Service (Gjovik, 1979) and by a manufacturer (Linsey, 1979). The potential for release from trucks remains; however it was not possible to estimate the contribution due to transport. Accidental spills at points of origin or destination or while the material is enroute can occur, but to date no such incidents have been documented.

2. Wood Preserving

The wood preserving industry consumes more pentachlorophenol than all other consumers combined. PCP is used for cosmetic reasons to prevent discoloration, to enhance toughness, and to prevent attack by wood-destroying fungi and insects. Precise estimates of consumption vary from year to year and depend upon the source (timber industry, manufacturer, federal government, etc.), but all are in the same range: Mannsville Chemical Products reports 78% in 1976 (Versar, 1978); U.S. EPA (1974) reported 78% in general; manufacturers project as high as 85% to 93% in 1978 (Linsey, 1979).

The timber industry has reported to EPA that 415 wood preserving plants are currently operated in the United States by approximately 300 companies (44 FR 62810-62846). Plants are geographically located in a manner consistent with the natural timber range, with heaviest concentrations in the Midsouth, Southeast and Pacific Northwest as shown in Figure 4. Wood preserving involves a two-stage process: first the wood is conditioned to reduce natural moisture content, thereby increasing permeability; second, the resulting wood is impregnated with PCP. EPA has designated the first stage as the most significant wood preserving-related contributor to wastewater flows (44 FR 62810-62846).

Two wood conditioning procedures predominate in the United States, which, when PCP is used, result in high phenolic concentrations in water. The Boulton conditioning process heats wood immersed in an oily, acidic (pH of 4-6) preservative inside of a treatment cylinder. The steam conditioning process steam pressurizes wood, also in a treatment cylinder, and this is followed by a vacuum cycle to reduce and remove moisture.



Total: 18,000 Metric Tons

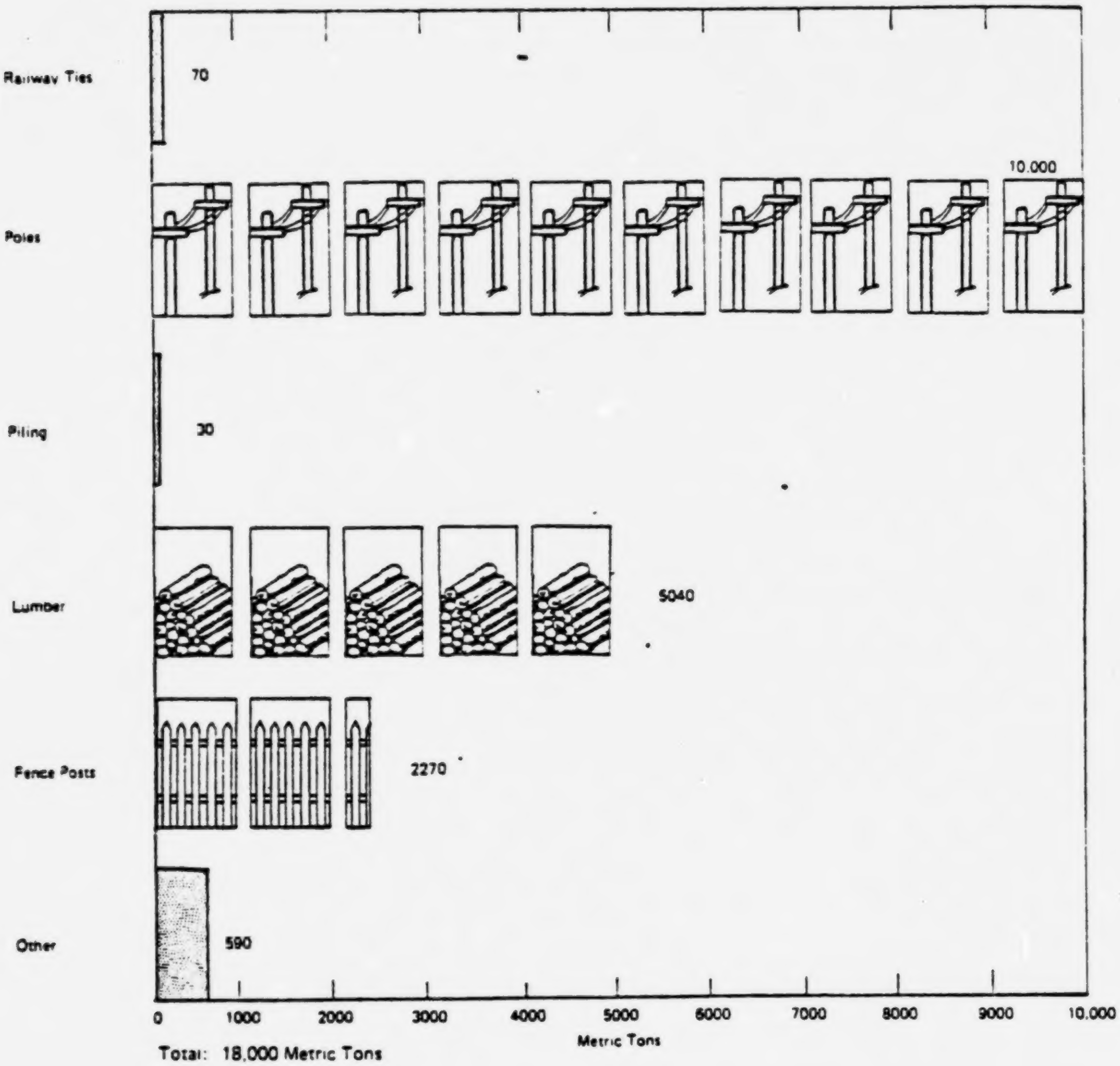
Source: Arthur D. Little,Inc., and American Wood-Preservers' Association (1975) and U.S. International Trade Commission (1979).

FIGURE 4 REGIONAL CONSUMPTION OF PENTACHLOROPHENOL BY WOOD PRESERVATION PLANTS (Estimated 1978)

Approximately 90% of existing wood treatment plants evaporate their wastewaters and, consequently, have no discharge to water at all. EPA reports that 11 plants utilizing the Boulton process and 31 steam plants introduce discharge to POTW's, as allowed by EPA guidelines. (These regulations are currently being revised.) Only one plant discharges directly to water on an intermittent basis, at a rate between 10,000 and 14,000 gallons per day; with concentrations of PCP between 10 and 20 mg/l the total release is estimated to be 3.8 to 26.5 kg annually for 10 to 25 operating days each year. (EPA, proposed, 1979). In the Development Document for Effluent Limitations Guidelines and Standards-Timber Products (proposed, 1979), EPA reports sampling results from 65% of the steam process indirect discharges and 25% of the Boulton process indirect dischargers. The efficiency of wastewater treatment varies; these data indicate that steam conditioners treated wastewaters with about 81% efficiency and Boulton conditioners with about 44% (the Boulton had a lower pretreatment concentration of PCP). Associated discharges to POTW's by steam and Boulton were, respectively, 5.1 and 0.2 MT of PCP annually (44 FR 62810-62846).

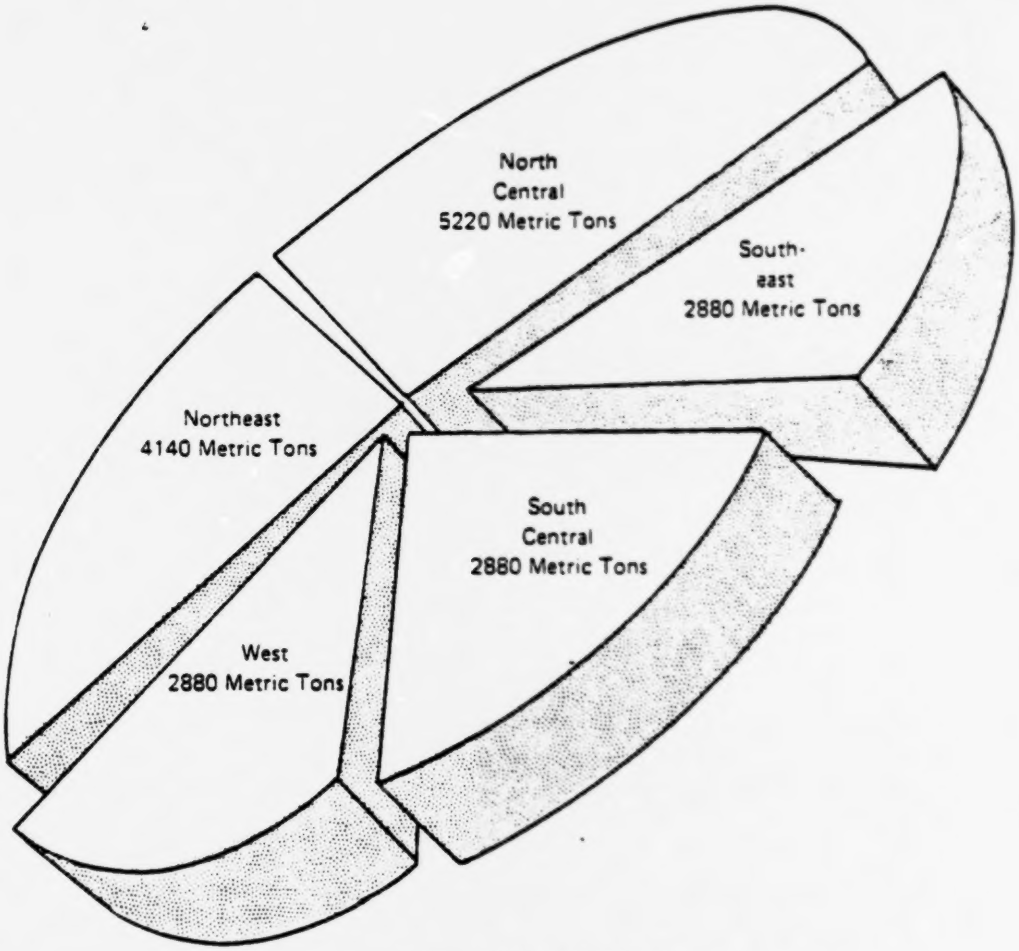
The majority of constituents removed by wastewater treatment probably accumulate in sludge. Though inconsistencies exist in sludge data and in the sludge practices themselves, the quantity of PCP in sludge from wood preservation can be estimated roughly. Approximately 14.3 MT of PCP sludge are generated per year by indirect dischargers, for treatment efficiencies as discussed above (EPA, 1979). The remainder of the industry, with no discharge, produces from the evaporation stage solid waste containing roughly 60.2 MT of PCP annually (EPA, 1979 and Arthur D. Little, Inc., estimates 1979). PCP deposited in sludge at industrial sites is largely believed to remain there (Arthur D. Little, Inc.); although it decays fairly rapidly in the presence of light, oxygen and microorganisms (see Section IV-C), conditions conducive to degradation cannot be assumed to exist at all sludge disposal sites.

Preserved wood is consumed by a variety of applications and is more evenly distributed geographically than are wood preservation plants. PCP is applied to wood at the widely published rate of about 0.23 kg PCP per cubic foot of wood (Rao, 1978). A distribution of wood products treated with PCP is presented in Figure 5, and regional consumption of PCP-treated wood is shown in Figure 6. By far, the largest application is to poles, comprising 57% of the total PCP consumption by the timber industry. Other applications include fenceposts (13%) and railroad ties (1.4%). Since these treated products are used out of doors the treated wood is exposed to environmental conditions (sunlight, rainfall, etc.). It was not possible to estimate the amount of PCP lost from treated wood through leaching and runoff. These sources contribute non-point releases to land, water and POTW's. Approximately 344 MT of PCP is volatilized from treated wood to the air annually (Arthur D. Little, Inc., estimates). Assuming that wood consumption is associated with land area, as illustrated in Figure 6, PCP release from treated wood by the previously described processes is expected to be distributed fairly evenly throughout the regions of the U.S.



Source: Arthur D. Little, Inc., American Wood-Preservers' Association (1975)., and U.S. Forest Service (1979).

FIGURE 5 MATERIALS TREATED WITH PENTACHLOROPHENOL
(Estimated 1978)



Total: 18,000 Metric Tons

Source: Arthur D. Little, Inc.; American Wood–Preserver's Association (1975); U.S. International Trade Commission (1979).

FIGURE 6 U.S. CONSUMPTION OF WOOD TREATED WITH PENTACHLOROPHENOL BY REGION (Estimated 1978)

Another source of release of PCP would be to the atmosphere through open-burning of treated wood. This is most likely to occur after some period of exposure of the wood to environmental conditions so that some portion of the original PCP will have already been lost through volatilization and leaching. Based on calculations described in Section IV-D, an estimate of 26 MT of PCP annually is expected to enter the atmosphere from burning of treated wood products.

3. Sodium Pentachlorophenate

Roughly 9% of the pentachlorophenol produced in the United States is used in the manufacture of sodium pentachlorophenate. This material is generally consumed as a fungicide and bactericide. The three most significant applications are in water cooling towers and as a preservative in paint and adhesives; each is discussed briefly below.

4. Other Uses of Pentachlorophenol and Sodium-PCP

Although the wood preserving industry consumes as much as 90% of PCP produced, there are additional applications which, because of their nature, are likely to result in human exposure and, therefore, merit discussion.

a. Paints

Pentachlorophenol is added to outdoor paints to enhance the water resistance of items such as porch furniture, trailers, boats, and outdoor trim. Occasionally these paints are misused and applied indoors (see Section V-B). The paint industry reports consumption of roughly 800 MT of PCP annually (Linsey, 1979).

Information has been published indicating that PCP may be used in toy paints (van Langeveld, 1975). Within the domestic toy industry, procedures have been tested and guidelines established to ensure that negligible amounts of PCP are used in toy manufacture or are contained in the final product (Arthur D. Little, Inc., estimates, 1979). However, van Langeveld (1975) has reported the results of a sampling of 65 commercial toy paints in Europe; nine were found to contain PCP in concentrations of 0.01%-0.27% (detection limit 4 mg/kg). The types of water colors and finger paints tested are imported into the U.S. Some manufacturing for U.S. toy companies is contracted out to foreign operators. Some of the toys may also contain paint to which PCP has been added. At present, data are insufficient to estimate the volume of PCP involved in these applications and the magnitude of any resulting releases to the environment.

b. Cooling Water Towers

Cooling waters used in industrial complexes are warm and well oxygenated, conditions favorable to growth of microorganisms. When microorganisms do grow, they promote equipment degradation, decrease operating efficiency and product quality, and present a potential health hazard. Proliferation of microorganisms is induced by factors both internal (system design, temperature, pH, and process materials) and external (water supply quality, site geography, and nature of aerosol contaminants) to the system (MITRE, 1979). Industrial cooling systems are decreasingly using adjacent stream waters due to environmental concern over thermal pollution of that media. Recirculating cooling water networks (especially cooling water towers) are replacing them. In addition to industrial applications, cooling water systems are also commonly found commercially in air conditioning systems in office buildings and large-scale computer operations (MITRE, 1979).

PCP is used in cooling water systems in two ways, each providing potential environmental release of the material. First, wooden slats, treated with PCP, are placed in a grid system to break the fall of cooling water into droplets and to optimize the heat exchange with the environment. The quantity of PCP released from this application is expected to be minor compared with other cooling tower uses.

Secondly, application of PCP to cooling waters themselves was estimated to be 230 metric tons in 1978 (MITRE, 1979). Recirculating Na-PCP-treated water has a greater potential for environmental release than PCP-treated wood slats. After passing through the industrial system which increases its temperature, water is depressurized, returned to the cooling tower and cooled as a result of its exposure to air. During temperature reduction of the cooling waters, vapor is emitted to the atmosphere, potentially containing PCP. There are data indicating that a high percentage of PCP consumed by this application is released to the air in this fashion. (Conservative estimates would assume that ultimately all PCP and the waters containing it are emitted to the atmosphere except that which is discharged during a system washout.) A more thorough investigation into this type of release is recommended.

Cooling water systems are occasionally washed out to clean the system and combat the build up of salt and other impurities. Typically these waters are treated before discharge. Based upon EPA-supplied effluent data (as yet unpublished) from industrial sites using cooling towers, it is estimated that 2 MT of PCP is discharged to water by this use, and the remaining 228 metric tons to air. If the aquatic discharge is treated, roughly 75% would be deposited in sludge; however, the nature of treatment practices is poorly documented.

c. Tanning

Pentachlorophenol and sodium pentachlorophenate are frequently combined with 2,4,5-Trichlorophenol (TCP) during product formulations in the leather industry (MITRE, 1979). Tanning is the chemical process during which natural hides and skins are converted to stable, durable leather. Because of its natural condition before treatment, leather is susceptible to infestation by bacteria molds and yeasts; consequently, PCP is used to protect the material during extended exposure to moisture.

Rarely is PCP used by itself. In most instances, PCP supplements the application of 2,4,5-TCP because PCP is less expensive and the use of these chemicals together is more effective. Annual consumption of 2,4,5-TCP is more substantial--approximately five times as great as that of PCP (MITRE, 1979).

On the basis of a survey of approximately 43% of the industry, it is estimated that approximately 15 MT of PCP is consumed annually (MITRE, 1979); this amounts to less than 0.1% of annual PCP production.

The use of PCP and its sodium salts in the tanning industry is expected to decline (MITRE, 1979). Particularly at early stages of treatment in which PCP is applied, improvements have speeded up the process, decreasing the time the leather is vulnerable to fungal growth; as a consequence, the MITRE Corporation (1979) suggests that PCP is not critical for tanning treatments.

Limited monitoring data on tanning industry effluents expected to become available in the near future would enable rough projections of PCP discharge directly to waters and/or indirectly to POTW's (EPA, 1979). At this time, however, only conservative estimates are possible based on knowledge of the application procedure. Preservatives are introduced in the early stages of tanning (batch soaking), as discussed above, and in finishing (sprays, pastes). PCP is used more frequently in the former process; if 80% is used in any of several soakings and 30% of the PCP in solution is absorbed or decays, then the remainder, approximately 8 MT per year, is discharged and treated at an assumed efficiency of 75%. Therefore, about 6 MT of PCP is released in sludge to to land and 2 MT to waters (MITRE, 1979).

In general, use of leather protectors decreases in winter months when fungi are less likely to abound.

d. Textiles

As is the case for many other uses of PCP, the chemical controls fungi and rot fungi when introduced to textiles and is registered with the EPA for this use (MITRE, 1979). In a study of this industry, MITRE indicates that all identified users add sodium-PCP to finishing solutions at concentrations ranging from 0.25% to 0.31% (MITRE, 1979). The total PCP and sodium-PCP consumed by the industry is about 20 MT annually. Roughly 60% or 12 MT of this is discharged for treatment (MITRE, 1979). Sixty-five percent of the textile plants have biological treatment with a 70% efficiency based on BOD removal (U.S. EPA 1980; MITRE 1979). Therefore 5.5 MT of PCP is discharged to surface water annually.

e. Other Uses and Releases

The most significant consumptive uses and sources of environmental release that can be identified and quantified given the current state of knowledge have been discussed in the preceding sections. However, several additional uses and potential sources of release should be noted. These are relatively minor applications and little is known of their precise nature at this time, although future studies in these areas may be appropriate.

PCP is registered with the EPA for use as a disinfectant, for application to masonry, agricultural seeds, paper mill systems, and construction materials such as: asbestos shingle, roof tiles, brick walls, concrete blocks insulation and pipe sealing compound (Rao, 1978). The petroleum industry has injected PCP into drilling muds, gypsum muds and packer fluids. It is added to photographic solutions to control slime and fungus (Rao, 1978). PCP is used at concentrations of 15-40 mg/l in secondary oil recovery to control microbial growth and optimize recovery (Rao, 1978).

In Europe, hexachlorobenzene (HCB) is used as a predecessor to PCP by manufacturers. Because of similarities in the chemical nature of PCP and HCB, biotransformation of HCB to PCP occurs with relative ease. Because of this, some residues of PCP may be attributable to HCB pollution (Conklin and Fox, 1978).

In the past, PCP has been used as an herbicide, both commercially and in home and garden applications. The U.S. Department of Agriculture reports its use by commercial mushroom houses to prevent proliferation of undesirable microbial life (MRI, 1974). It has been used on non-crops and dormant crops during pre-harvest periods. Approximately 200 MT per year is consumed in this fashion (Versar, 1978). The material can be bought in retail hardware stores for use by homeowners as a bird repellent and garden insecticide. Approximately 600 MT is consumed annually by homeowners. Both of these applications are assumed to be direct releases to the environment as non-point and point sources.

Pentachlorophenol is used as a preservative in many food-associated products such as adhesives on food packaging, coatings on paper and paperboard used in food producing, manufacturing, packaging, processing, preparing, treating and transporting. Consequently, the use of PCP in this area is covered by the Food Additives regulations set forth in Title 21 of the U.S. Code Annotated. A summary of potential PCP applications is presented in Table 3; where limitations have been articulated, they are included.

5. Investigations of POTW's

There are five known sources of PCP release to sewer systems that will reach POTW's: PCP production plants, textile factories, the leather tanning industry, approximately 10% of the wood preserving facilities, and cooling towers. The amount of PCP waste discharged to sewers that has been accounted for is 9 MT per year from these sources. The wood preserving industry reports a total discharge of 6 MT per year from only 42 plants of the total 415 operating facilities. An additional 3 MT per year is associated with leather tanning plants. The discharges from the

TABLE 3. SUMMARY OF FDA-APPROVED USES OF PENTACHLOROPHENOL IN FOOD-ASSOCIATED PRODUCTS

Product Description	Substances Used	Recommend Concentrations/ Limitations (where available)
Paperboard products used in food packaging	Potassium - PCP Sodium - PCP	
Preservative for ammonium alginate used in manufacture of PVC emulsion polymers	Sodium - PCP	≤ 0.5% by weight
Preservative for wood used in packaging/transport raw agricultural products	PCP: Sodium - PCP	≤ 50 mg/kg/PCP
Animal glue	Potassium - PCP Sodium - PCP	Only as a preservative
Textiles used in manufacturing, processing, packaging, transport, etc. of food	Sodium - PCP	Only as a preservative
Rubber articles used in manufacturing, processing, packaging transport, etc. of food	Sodium - PCP	≤ 5.0% by weight
Closures with sealing gaskets on containers for food	Potassium - PCP Sodium - PCP	≤ 0.05% by weight of closure sealing gasket
Slimicides used in manufacture of paper (board) that will contact food	Potassium - PCP Sodium - PCP	
Defoaming agents used in manufacture of paper (board) that will contact food	Potassium - PCP Sodium - PCP	
Defoaming agents used in manufacture of coatings for articles that will contact food	Potassium - PCP Sodium - PCP	Only as a preservative
Uncoated/coated food-contact surface of paper (board) in packaging, etc.	Sodium - PCP	Only as a preservative
Resins and polymeric coatings used as food contact surface in manufacturing, packaging, transport, etc.	Sodium - PCP	0.1% by weight in can-sealing compounds on containers with ≥ gallon capacity.
Adhesives of articles used in packaging transport, etc. of food	PCP, Sodium - PCP Potassium - PCP	Only as a preservative

[1] The above were taken from various portions of the Food Additive regulations of the Food and Drug Administration, Title 21, U.S. Code.

three remaining sources have not been quantified to date, but it is expected that production-associated wastes are probably negligible.

Total loadings may be estimated by a doubling of the known discharge to sewers of 9 MT to 18 MT per year. This accounts for contributions from textile works and cooling towers, as well as runoff from PCP-treated outdoor wood products which enters sewage systems in runoff. A total of 18 MT is conservatively estimated to account for all unquantified sources of PCP.

Most known sources of PCP to sewers are localized. The wood preserving industry is concentrated in the eastern half of the United States, especially the Southeast and the Northwest. Discharges from other sources, such as textile facilities, tanning plants and cooling towers, are intermittent point sources and more widely distributed. Release of PCP from treated wood products is expected to be distributed evenly across the U.S. The amount of PCP that would reach storm sewers from this source relative to other sources is unknown so it is not possible to rule out an even distribution of PCP on a national basis. However, based on the assumption that the wood preservers are significant dischargers to POTW's, any estimations of total sewage PCP loading based upon effluent concentrations and total U.S. POTW flow will be too high or low, depending on the location where the effluents were sampled.

The following sample calculation uses the estimation procedure of Versar (1979) but with revised numbers. Table 4 presents PCP concentrations detected in POTW final effluents in several cities. Excluding the Grand Rapids measurement as an outlyer, a mean concentration of 10.5 µg/l can be calculated. Then the calculation, based on a total flow in U.S. POTW'S of 26,205 million gallons per day (EPA, 1978) follows:

Total PCP in sewage effluent = average concentration x flow = (10.5 µg/l) x (365 days/year) x (26,205 x 10^6 gal/day) x (3.785 l x 10^{-12} MT/µg) = 380.13 MT

This estimate may be too high due to assumptions of the model such as using daily flow estimates for 365 days per year. Another factor is the small amount of data supporting the estimated average effluent concentration. Most of the POTW's sampled in Table 4 are located in states with numerous wood preserving industries which may contribute to effluent concentrations higher than the national average. Without more data, however, no firm conclusions can be made concerning the contribution of POTW discharges to surface water concentrations of PCP. In Table 2, 18 MT describes the amount of PCP contributed to the environment by POTW sludge. Three hundred eighty MT approximates the amount of PCP discharged to water in POTW effluents. The discrepancy between the numbers implies either:

(1) a low efficiency for PCP removal from water onto sludge, assuming limited disappearance in the system or

TABLE 4. PCP CONCENTRATIONS IN POTW FINAL EFFLUENT[1]

City	Concentration (μg/l)	Number of Days Days Sampled
Indianapolis, Indiana	1	12
Cincinnati, Ohio	1	7
Lewiston, Maine	< 50	4
Atlanta, Georgia	< 5	6
St. Louis, Missouri	< 5	6
Pottstown, Pennsylvania	1	6
Grand Rapids, Michigan	<250[2]	6

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[1] Preliminary data to be verified from Burns and Roe POTW study sponsored by Effluent Guidelines Division.

[2] Effluent before chlorination.

(2) a high efficiency for PCP degradation in POTW sludge, coupled with a very large combined industrial discharge to POTW's (>>400 MT) leaving only a small fraction behind in sludge.

Assuming a total POTW efficiency of 80%[1] (which includes removal at all treatment levels), then 20% of the total amount of PCP discharged by industries to POTW's equals the amount leaving in POTW effluents (380 MT) and sludge (18 MT), totalling 400 MT of PCP annually. From this number the total industrial input to POTW's is estimated at 2000 MT annually. Due to the large number of assumptions made in deriving this POTW loading estimate, it was not used as input to the materials balance of PCP in Table 2.

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[1] Based on observations in wastewater treatment and POTW treatment of efficiencies of ≈100% and 60% averaged (see Sections IV-B-2 and IV-C-1-c-vi). Excluded from this average was a low efficiency of 4-28% (observed in one treatment plant) because it was not a typically observed number, it was derived from a small sample and on evidence of biodegradation of PCP in laboratory environments.

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SECTION IV.

DISTRIBUTION OF PENTACHLOROPHENOL IN THE ENVIRONMENT

A. INTRODUCTION

This section of the exposure assessment describes the distribution of PCP among different environmental media following release from its sources. The section contains a discussion of:

(1) monitoring data presenting concentrations of PCP measured in various media,

(2) physico-chemical fate processes that determine these PCP concentrations through transformation or transport,

(3) biological fate processes, and

(4) concentration estimates for several critical environmental pathways.

The overall approach chosen to analyze PCP's environmental distribution was determined by the nature of the chemical's entry into the environment, its physico-chemical properties, and the availability of data. The majority of the pollutant loading of the environment with PCP does not enter through direct discharge but from numerous, often unknown, non point sources. No particular environmental compartment stands out as the primary recipient of PCP release, and transfer of the pollutant between compartments appears to occur. PCP is relatively persistent in most media, based on its physico-chemical properties, which suggests that physical transport is equally or more important than chemical transformation. Finally, lack of monitoring data necessitates estimation of PCP environmental concentrations for use in the exposure sections. Because fate data are so limited, these estimates are based on simple calculational models and many assumptions in some cases.

Several approaches have been described for the environmental fate and pathways analyses required for an exposure assessment (ADL, 1980). The critical pathways approach was used for PCP, making use of estimated pollutant environmental loadings to different environmental media based on materials balance data. Information on fate was reviewed in the first section and used, when applicable, in the estimation of environmental concentrations. The approach was limited by data gaps concerning loadings to certain compartments, (e.g., water) on a national scale and for the most part, did not address intercompartmental transfers. The results of this analysis are not inclusive, therefore, nor do they account for the total mass of PCP entering the environment. The analysis was focused on what appeared to be the important pathways, largely because of the availability of data required for estimation. Whenever possible, monitoring data were used to verify the results of the estimations.

B. MONITORING DATA

1. Introduction

Monitoring data giving PCP concentrations in various environmental media are too few to be systematic and indicative of the national distribution of the chemical. Data are scattered, inconsistent and primarily anecdotal. At best, the information that is available can be used to verify estimates of environmental concentrations based on fate models. The following section presents, by environmental medium, the available monitoring data.

2. Water and Sediment

Tables 5 through 10 summarize levels of PCP found in water and sediment over the last 10 years as reported in the literature. Monitoring data available through STORET were found to be very limited, with a total of 497 observations, 82% of which were remarked data (at the detection limit) as of March, 1979; for the unremarked data (above detection limits), 84% of the observations fell between 0.1 μg/l and 10 μg/l, with a total range of 0.01 μg/l to 100 μg/l. For unremarked and remarked data combined, the majority of non-zero observations (approximately 55%) fell between 0.1 μg/l and 100 μg/l, with all values less than 100 mg/l.

The data imply that ambient levels of PCP in surface water are usually below 1 μg/l, with much higher levels in more industrialized areas. Bevenue et al. (1972b) analyzed samples of snow, rainwater, and lake water in Hawaii where PCP is used extensively for wood preservation and termite control (see Table 6). The authors attributed the consistent presence of PCP in the area mainly to the use of construction lumber treated with preservatives containing PCP.

Young et al. (1975) found results comparable to those of Benevue in streams feeding into the Kaneohe in Hawaii Bay and in the Bay itself (see Table 6). Mean values for the streams were 0.12 μg/l and for the Bay 0.07 μg/l. Values for samples taken near sewage outfalls in the Bay (0.068 μg/l) did not vary significantly from values for samples collected at other stations (0.073 μg/l). In the Netherlands, Wegman (1979) reports PCP levels in five major rivers averaging 0.49 μg/l in 1976 and 0.93 μg/l in 1977 (see Table 7).

Preliminary data on PCP concentrations in various POTW treatment stages are presented in Table 8. Final effluent concentrations varied from 1 μg/l to <50 μg/l. Levels in the influent fell within the same range: from none detected to <50 μg/l. It was not possible to calculate an average treatment efficiency due to differences in treatment methods and analytical techniques between POTW plants. For the Pottstown and Atlanta plants, however, efficiencies calculated were 93% and 100%, respectively. A study of PCP content in the sewage of three Oregon cities conducted by Buhler et al. (1973) found the average percentage of PCP removed at the treatment plants to be 59%. POTW influent ranged

TABLE 5. TOTAL PENTACHLOROPHENOL IN AMBIENT WATERS

(Remarked and Unremarked Data)

Region	Percentage of Observations					
	0.001-0.01 μg/l	0.01-0.1 μg/l	0.1-1 μg/l	1-10 μg/l	10-100 μg/l	No.
New England	-	-	-	-	-	0
Mid Atlantic	-	-	-	-	-	0
Southeast	-	-	-	-	-	0
Great Lakes	0	0	100	0	0	8
Ohio	0	0	0	100	0	6
Tennessee	-	-	-	-	-	0
Upper Mississippi	-	-	-	-	-	0
Lower Mississippi	-	-	-	-	-	0
Souris and Red of North	-	-	-	-	-	0
Missouri	0	0	0	67	33	3
Arkansas and Red	-	-	-	-	-	0
Western Gulf	-	-	-	-	-	0
Rio Grande and Pecos	-	-	-	-	-	0
Upper Colorado	-	-	-	-	-	0
Lower Colorado	-	-	-	-	-	0
Great Basin	-	-	-	-	-	0
Pacific Northwest	9	0	0	0	91	22
California	0	32	17	44	7	41
Alaska	-	-	-	-	-	0
Hawaii	-	-	-	-	-	0
United States	3	16	19	33	30	80

Source: STORET: Data from 6/78-6/79.

TABLE 6. MEAN CONCENTRATIONS OF PCP IN WATER

Site/Type of Sample	No. Samples	Concentration (μg/l) Mean	Range	Source
Drinking water	108	0.07		NOMS[1]
River below papermill		9		Rudling (1970)[2]
Lake below papermill		3		Rudling (1970)
Riverwater below wood preserving plant		0.35		Renberg (1974)
Streams (Hawaii - Kaneohe)	63	0.12	0.01-0.48	Young *et al*. (1976)
Bays (Hawaii - Kaneohe)	29	0.07	ND[3] -0.38	Young *et al*. (1976)
Rainwater (Hawaii)				Bevenue *et al*. (1972b)
Kailua		0.02		Bevenue *et al*. (1972b)
Kaneohe		0.012	0.016-0.008	Bevenue *et al*. (1972b)
Waipahu		0.014		Bevenue *et al*. (1972b)
Honolulu		0.1	0.002-0.284	Bevenue *et al*. (1972b)
Snow		0.014		Bevenue *et al*. (1972b)
Lake Water		0.01		Bevenue *et al*. (1972b)
Sewage influent (Oregon)		4.3	1-5	Buhler *et al*. (1973)
Sewage effluent (Oregon)			1-4	Buhler *et al*. (1973)[4]
Willamette River water (Oregon)			0.1-0.7	Buhler *et al*. (1973)
Drinking water for Corvallis			0.04-0.28	Buhler *et al*. (1973)
Kalihi stream (industrial area - Hawaii)		0.655		Bevenue *et al*. (1972a)
Sand Island outfall (sewage - Hawaii)		2.6		Bevenue *et al*. (1972a)

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[1] NOMS = National Organics Monitoring Survey.

[2] Recovery = 94%.

[3] ND = non detectable.

[4] Average recovery = 103%.

TABLE 7. PCP IN SURFACE WATERS OF THE NETHERLANDS

Site	Concentration (μg/l)			
	1976		1977	
	max.	med.	max.	med.
Rhine	2.4	0.73	11	1.1
Boven Mernede	2.6	0.49	9.6	1.0
Ijssel	2.00	0.55	10	1.0
Meuse	3.9	0.38	8.9	0.79
Meuse	1.4	0.32	10	0.78
		0.494		0.934

Detection limit: 0.01 ng/l

Recoveries >85%

Source: Wegman (1979)

TABLE 8. AVERAGE CONCENTRATIONS OF PENTACHLOROPHENOL IN POTW'S[1]

Type of Sample	Indianapolis	Cincinnati	Lewiston	Atlanta	St. Louis	Pottstown	Gr. Rapids	Flint	Hartford
Influent	1	1	<50	6	<5	15	<27	<18	ND
Effluent Pre Chlorination	3	2	<50				<250		
Final Effluent	1	1	<50	5	<5	1			
% Removal				100		93			
Primary Sludge	93		ND		ND	ND			
Waste Activated Sludge	112								
Flotables	1								
Combined Sludge		50	30	25			1000		
Gravity Thickened Overflow			<50						
Digested Sludge				ND	5	ND			
Heat-treated Sludge							ND		
Heat Treatment Decant							<250	60	
Secondary Effluent (Chlorinated)								<12	ND
Number of Days Sampled	12	7	4	6	6	6	6	NA	NA

[1] Average concentration in μg/l.

Source: Preliminary data from Burns and Roe POTW Studies for Effluent Guidelines Division U.S. Environmental Protection Agency.

TABLE 9. PCP IN WATER AT VARIOUS LOCATIONS

Location/Type of Sample	Mean Concentration in 24-hr. Samples (µg/l)		Output g/10000 pop., 24 hr.
	Influent	Effluent	
Corvallis Sewage Plant[1]			
Corvallis	1.4	1.0	5.5
Eugene	4.1	3.3	26.0
Salem	4.6	4.4	33.6

Non-Potable Water, Hawaii[2]	Concentration (µg/l)	
	Mean	Range
Water (industrial area)	0.655	0.168 - 1.143
Sewage	2.6	

Points on Naylors Creek[3]	Concentration (µg/l)	
	Mean	Range
Culvert (factory area)	6171	82 - 10,500
Manoa Rd.	179	120 - 240
Manor Rd.	150	71 - 230
Garrett Rd.	43	38 - 49
Oil sample at factory outlet	5.38×10^6	$5.3 \times 10^6 - 5.45 \times 10^6$

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[1] Buhler *et al.* (1973)
[2] Bevenue *et al.* (1972a)
[3] Fountaine *et al.* (1975)

TABLE 10. PCP IN WATER AND SEDIMENT[1] OF FRESHWATER ECOSYSTEM

Sample Date	Water(μg/l) Mean	Water(μg/l) Range	Sediment (dry)(μg/kg) Mean	Sediment (dry)(μg/kg) Range	Leaf Litter (dry) Mean(μg/kg)
2/27/75[2]	10.3	6-19	162.2	2-800	6400 ± 250
4/24/75	7.8	6-15	254.8	21-1160	2550 ± 650
6/28/75	18.3	2-82	474.2	5-1300	5200 ± 300
8/05/75	2.25	1-3	116	10-207	
10/11/75	2.8	<1-10	269.5	24-927	5800 ± 1200
12/6/75	21.2	1-76	253.6	11-900	3470
2/7/76	18	10-29	117.6	10-313	1680 ± 12
5/3/76	6	2-18	36	7-96	6000 ± 1000
8/11/76[3]	2.8	0.1-11	487	142-857	
10/22/76	0.5	2.3-1	440.3	166-994	
1/5/77	36.5	16-81	209	250-277	
2/22/77	88	29-147	1518	1518	
4/27/77	7.8	5-16	156.8	4-250	

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[1] Six sites on the lake and inlet stream were sampled.

[2]

	Recovery of PCP	Background levels in control pond
water	90 ± 15%	0.5 μg/l
sediment	90 ± 38%	5 μg/kg
leaf litter	62 ± 4%	50 μg/kg

[3]

water	95 10%	0.28 μg/l
sediment	113 25%	3.3 μg/kg

SOURCE: Pierce et al. (1977) and Pierce and Victor (1978).

from 1 μg/l to 5 μg/l PCP, effluent ranged from 1 μg/l to 4 μg/l PCP, and the Willamette River samples ranged from 0.1 μg/l to 0.7 μg/l PCP. Bevenue et al. (1972a) reported PCP levels of 2.6 μg/l at the Sand Island outfall in Hawaii (see Table 9).

Fountaine et al. (1975) studied the effects of untreated industrial effluents in a stream running through an industrial district with several manufacturing (including wood-preserving) plants in Pennsylvania; they found PCP levels ranging from 0.038 mg/l to 10.5 mg/l (see Table 9). The authors believe that their findings result from direct discharge into the stream, with the additional possibility of chronic seepage from PCP-saturated soil in the factory area.

Similar measurements of industrial waste outputs have been reported by Pierce et al. (1977 and 1978) (see Table 10). PCP levels in a stream and lake below a wood preserving plant in Hattiesburg, Mississippi, were studied over a 2-year period, during which time two fish kills linked with PCP levels occurred in the lake. Fluctuations in the mean PCP levels of the water--from 0.5 μg/l to 88 μg/l--have been attributed to either periodic overflows into the stream from the plant's holding pond or leaching from the contaminated watershed. Of greater concern were consistently high levels in the sediments, an average over 2 years of 650 μg/kg, giving rise to chronic pollution of the ecosystem.

3. Humans

Data have been collected on PCP levels in human urine and tissue for both occupational and non-occupational populations (see Tables 11 and 12). While levels are significantly higher in occupationally exposed groups, tests on the general population consistently show evidence of low-level exposure. In an FDA study in Florida, Cranmer and Freal (1970) found average PCP urine levels of 4.9 μg/l in the general population compared with 119.9 μg/l in carpenters, boat builders, and spraymen. Bevenue et al. (1967a) cite a range of 1100-5910 μg/l in the urine of Japanese pest control operators exposed to PCP compared with 10-50 μg/l in non-exposed workers.

Bevenue et al. (1967b) compared results from a study in Hawaii on PCP in the urine of three groups; occupational, non-occupational and a mixed population. The occupational population's level of 1802 μg/l is almost 50 times higher than the non-occupational group's level of 40 μg/l. This is comparable in magnitude to the differences noted above in the studies in Florida and Japan. The results of Kutz et al. (1978) agree with those of the Florida study: a mean level of 6.3 μg/l in 418 urine samples collected from the general U.S. population. Shafik (1973) found a mean value of 26.3 μg/kg in adipose tissue from the general U.S. population and concludes, "humans are continuously exposed to low levels of PCP and HCP from the environment, food supplies and disinfectants."

TABLE 11. PCP LEVELS IN HUMAN TISSUE AND URINE

Population and Sample	Concentration (μg/kg and μg/l) Mean	Concentration (μg/kg and μg/l) Range	Reference
Exposed workers - urine (Japan)		1100-5910	Bevenue (1967a)
Non-exposed workers - urine (Japan)		10-50	Bevenue (1967a)
General population - urine (Florida)	4.9	2.2-11.2	Cranmer (1970)
Occupational workers - urine (Florida)	119.9	22.2-270	Cranmer (1970)
General Population - adipose tissue	26.3	12-52	Shafik (1973)[1]
Occupational population - urine (Hawaii)	1802	3-35700	Bevenue (1967b)[2]
Non-occupational population - urine (Hawaii)	40	ND-1840	Bevenue (1967b)
Occupational/non-occupational population - urine	217	3-38642	Bevenue (1967b)
Combination of the above three groups (Hawaii)	587	ND-38642	Bevenue (1967b)
Occupational worker exposure - urine - by wood preserving methods (Oregon)			Arsenault (1976)[3]
Dip	2830	120-9680	
Spray	980	130-2580	
Pressure	1240	170-5570	
U.S. General Population - urine	6.3	ND-193	Kutz (1978)[4]

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[1] Detection limit = 5 μg/kg.

[2] Detection limit = 3 μg/l.

[3] Detection limit = 5 μg/l.

[4] Detection limit = 5-30 μg/l.

TABLE 12. PENTACHLOROPHENOL IN HUMAN URINE (FLORIDA)

Sample	Origin	Concentration μg/l Replicate Range[1]	Mean[2]
1	General Population	2.2 - 2.3	2.2
2	General Population	2.7 - 2.9	2.8
3	General Population	2.9 - 3.0	2.9
4	General Population	5.0 - 5.3	5.1
5	General Population	5.2 - 5.5	5.3
6	General Population	10.6 - 11.2	10.8
7	Carpenter	22.2 - 25.5	24.1
8	Boat Builder	55.0 - 60.1	57.3
9	Sprayman	131 - 136	133
10	Sprayman	259 - 270	265

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[1] Four replicates of each sample.

[2] Variation of the method was ±5%.

Source: Cranmer and Freal (1970).

In a study of workers exposed to PCP in the wood preserving industry, Arsenault (1976) reports PCP levels of 120-9680 μg/l in urine, with a mean of approximately 1683 μg/l. In a hospital where two infant deaths occurred due to PCP exposure, hospital diapers were found to contain an average of 61937 μg/l as a result of cleansing in a mildew preventative. Samples of serum of nurses handling the linen revealed a level of 5400 μg/l PCP.

4. Air

Limited information is available on the levels of PCP in air, although work by Bevenue et al. (1972a) has reported PCP levels of 0.002-0.284 μg/l in rainwater in Hawaii. In a study of 25 wood preserving companies in Oregon, Arsenault (1976) reported PCP levels in the air of the plants ranging from 0.003 mg/m^3 to 0.063 mg/m^3 (see Table 13). In Sweden, Levin and Nilson (1977) collected samples of air containing wood dust in trimming and grading plants, where boards treated with PCP were cut; with a recovery rate of 70%, they reported a mean value of 157,000 mg/m^3 PCP.

5. Food and Feed

Levels of PCP in food sold over the counter were examined as a part of FDA's ongoing Market Basket Studies (see Table 14). Heikes (1979) reported an average of 18 μg/kg with a range of 1.8-62 μg/kg, in peanut butter samples. The same study also reported finding PCP on the Mason jar lids used in home canning. Between August 1974 and July 1975, Johnson and Manske examined a wide range of products typically found in the diet of 16-19 year olds. The products were purchased at random locations, cooked, and then analyzed. PCP levels ranged from below detection levels to 40 μg/kg, and detectable amounts were found in dairy products, grain and cereal, leafy vegetables, root vegetables, garden fruits, fruits, and sugars and adjuncts. Grain and cereal, root vegetables, and sugars had the highest mean concentrations: 1, 1, and 6 μg/kg, respectively.

In Michigan in 1977 several dairy herds were quarantined because of the suspicion of possible PCP contamination from exposure to wood treated with PCP. Lamparski et al. (1978) analyzed milk collected from the herds and found no evidence of PCP content at detection limits of 10-15 μg/l.

6. Biota

The premise that the distribution of PCP in the aquatic environment is widespread would appear to be supported by reported levels of PCP (see Tables 15-17). Zitco et al. (1974) collected fish samples from the St. Croix and St. John estuaries and off the New Brunswick coast and bird eggs from White Horse and Hospital Island. PCP levels in biota from these relatively clean sample sites ranged from less than 0.5 ng/g to 10.83 ng/g wet weight and averaged 2.23 ng/g. Rudling (1970) examined fish in Sweden collected downstream from a pulp mill's effluent and

TABLE 13. PCP IN AIR AT OREGON WOOD PRESERVING PLANTS

Wood Preserving Process	Concentration (mg/m^3)	
	Mean	Range
Dip	.019	.003 - .063
Spray	.006	.003 - .012
Pressure	.014	.004 - .028

Source: Arsenault (1976).

TABLE 14. PCP IN FOOD AND FEED

Sample	Concentration (μg/l) and μg/kg) Mean	 Range	Reference
Dairy	0.5	10	Johnson and Manske (1977)[1]
Grain and Cereal	1	10 - 13	Johnson and Manske (1977)
Leaf vegetables	T	13	Johnson and Manske (1977)
Root vegetables	1	10	Johnson and Manske (1977)
Garden fruits	T	10	Johnson and Manske (1977)
Fruits	T	11	Johnson and Manske (1977)
Sugars	6	10 - 40	Johnson and Manske (1977)
Peanut butter	18	1.8 - 62	Heikes (1979)[2]
Bovine milk	ND		Lamparski *et al*. (1978)[3]

---

[1] T = average below detection limit. Samples collected through U.S. in FDA's Market Basket Study.

[2] Market Basket Study - U.S. population.

[3] Michigan dairy herds, detection level = 10 μg/l.

TABLE 15. PCP IN BIOTA

Sampling Site/Type	Concentration		Reference
	Mean	Range	
Lake (Sweden) - mg/kg			Rudling (1970)[1]
Pike	0.2		
Perch	0.15		
Eel	3.0		
Coastal Estuaries (New Brunswick, Canada) (ng/g wet weight)			Zitco (1974)
Cod	0.82		
Winter Flounder		1.77-3.99	
Sea Raven	2.5		
Silver Hake	1.75		
Atlantic Salmon		0.54-1.26	
White Shark Liver	10.83		
Double-Crested Cormorant Egg	0.36		
Herring Gull Egg	0.51		
Fish Food	2.23		

---

[1] Detection limit = .1 μg/kg.
Recovery:

Pike	87%
Perch	81%
Eel	91%

TABLE 16. PCP IN BIOTA

Lake (Mississippi)[1,2]

	Mean Concentration (ng/g dry weight)
2/27/75	2500 ± 200
4/24/75	1380 ± 20
6/23/75	130 ± 70
10/11/75	Trace
12/6/75	651 ± 650
2/10/76	87 ± 22
5/3/76	Trace

Recovery level = 62 ± 4%   Background level = 50 ng/g

	Muscle	Gills	Liver
10/11/76[3]			
Sunfish-1	5	N.A.[4]	26
Sunfish-2	4	N.A.	150
1/6/77			
Sunfish-1	9,400	48,400	130,000
Sunfish-2	6,400	N.A.	N.A.
Bass-1	7,000	42,000	200,000
Bass-2	17,000	N.A.	140,000
Bass-3	16,000	N.A.	325,000
Catfish-1	19,000	N.A.	214,000
4/27/77			
Sunfish-1	900	N.A.	14,600
Sunfish-2	1,000	N.A.	14,900
Catfish-1	8,200	N.A.	50,600
Catfish-2	1,500	N.A.	20,200

Recovery level = ± 8%

---

[1] Assumed to be combined values for sunfish, bass, catfish.

[2] Pierce *et al.* (1977).

[3] Pierce and Victor (1978)

[4] N.A. = not analyzed.

TABLE 17. PCP CONCENTRATIONS IN FISH TISSUE AS REPORTED IN THE STORET DATA BASE[1]

Major River Basin	Concentration (mg/kg) Range	Concentration (mg/kg) Mean	No. of Observations
Lake Michigan	0.002[2]	0.002	28
Lower Mississippi	0-1.3	0.478	48
Pacific Northwest	0.005-50.0[3]	16.38	33
Western Gulf	0	0	2
S. Central Lower Mississippi	0	0	1
Alaska	5.0	5.0	5

---

[1] For years 1976-79.

[2] All data are remarked.

[3] All data except for two observations are remarked.

found PCP levels ranging from 0.2 mg to 3.0 mg of PCP per kg of tissue. The STORET data base reports tissue concentrations ranging from 0 mg/kg to 50 mg/kg.

Pierce (1977 and 1978) studied a fresh water lake located below a wood treating plant in Mississippi from 1975 to 1977. Observations over this time period, which included two extensive fish kills, indicated periodic releases of PCP from the watershed. In the first study, PCP concentrations in fish ranged from 87 ng to 2500 ng/g dry weight tissue, while in leaf litter concentrations ranged from 1680 ng to 6400 ng PCP/g dry weight. In their second study, the authors reported ranges from a mean of 15.5 ng/g wet weight to a mean of 29400 ng/g wet weight tissue.

7. Soil

Although several authors refer to possible soil contamination as a source of PCP levels in water samples, very little data are available on actual measurements of PCP in soil. Fountaine (1975) says, "It is at least possible that some of the PCP discharge might not be a result of the present factory process but might be a result of the saturation of the factory area with excess PCP." Pierce et al.(1978) state, "The increase in PCP concentrations observed in February 1976 followed a period of heavy rainfall, indicating that PCP was leached from the contaminated watershed area..." Stark (1969) reported levels as high as 1.5% PCP on a contaminated lake shore in Sweden. The detection limit was .05 μg/kg with 80-100% recovery.

8. Miscellaneous Media

Von Langeveld (1975) examined PCP levels in the Netherlands in samples of children's paints (i.e., watercolors, finger paints) and reported levels ranging from 0.01 to 0.27 m/l in nine out of sixty-five samples. The detection limit was 4 mg/l.

C. ENVIRONMENTAL FATE

1. Physico-Chemical Fate Processes

a. Introduction

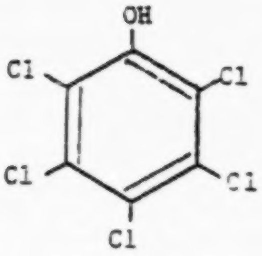
The following section describes, by environmental medium, the major physical and chemical fate processes affecting PCP concentration and distribution following its release. The media considered are air, water, soil and wood that has been treated with preservative. Table 18 presents some general chemical properties of PCP and lists the fate processes discussed in the following section for each medium.

b. Air

i. Atmospheric Photolysis

Very little information is available relating to the photolysis of PCP in the atmosphere. Two references (Gäb et al., 1975 and Crosby and

TABLE 18. CHEMICAL AND FATE PROPERTIES OF PENTACHLOROPHENOL



Chemical Structure of
Pentachlorophenol

Chemical Properties

Molecular Weight	266.35
Melting Point	190°C
Boiling Point at 760 torr	310°C
Vapor Pressure at 20°C	$1.1 - 1.7 \times 10^{-4}$ torr
Solubility in Water 20°C	14 mg/L
Acid Dissociation Constant	pKa = 4.74
Log octanol/water partition coefficient	5.01

SOURCE: Versar (1979).

Fate Processes

Air

- Photolysis
- Free Radical Oxidation
- Rainout

Water

- Hydrolysis
- Photolysis
- Oxidation
- Volatilization
- Biodegradation
- Adsorption/Sedimentation

Soil

- Sorption
- Leaching
- Hydrolysis
- Photolysis
- Volatilization
- Biodegradation

Treated Wood

- Photolysis
- Volatilization
- Leaching

Hamadmad, 1971) indicate that PCP is probably photolyzed in the absence of water, though the mechanisms for this are not well understood.

In one experiment (Gäb _et_ _al_., 1975), PCP was sorbed onto a silica gel or deposited as a film on a glass surface and then irradiated in the presence of an oxygen stream. "It was observed that the conversion rates of the substances absorbed on particulate matter were far higher than with those deposited as solids or thin films on a glass surface." This was attributed to a shift in absorption bands due to adsorption onto particles, as well as to the greater dispersion of pesticide molecules in the adsorbed phase resulting in greater contact of the pesticide with oxygen. CO_2 and HCl were formed during irradiation of PCP with wavelengths λ >290 nm. No values were given for the intensity or spectrum of light used; therefore it is not possible to relate the rates of degradation in the environment to those determined experimentally (Table 19).

TABLE 19. RESULTS OF PCP IRRADIATION IN UV LIGHT (λ >290 nm) UNDER TWO CONDITIONS

Condition	Irradiation Time (Days)	Initial Solid Material (mg)	Remaining Solid Material (mg)	Products (mg)[1]		
				CO_2	HCl	Cl_2
In oxygen stream	7	80	69	15	6	n.d.[2]
Adsorbed on silica gel		102				
	4		26			
	7		12	n.d.	n.d.	n.d.

[1] Other compounds could have been formed but not detected by analytical methods.

[2] n.d. = not detected.

Source: Gäb _et_ _al_., 1975.

In another experiment (Crosby and Hamadmad, 1971), PCP was dissolved at a concentration of 1 g/l in an organic solvent and irradiated at 25°C. The PCP solution turned pale yellow after exposure to sunlight for 7 days. When PCP was exposed as a film on glass, the same thing occurred. No decomposition products were detected by gas-liquid chromatography or thin-layer chromatography analysis. Similar PCP solutions were irradiated in the laboratory with a low-pressure mercury arc lamp radiating at 253.7 nm. (Note that this wavelenghth is below the atmospheric cutoff for solar radiation of ∿290 nm.) After 32 hours, 60% PCP, 30% tetrachlorophenol, and 10% of another phenolic substance were found. The experimenters report that "although definite photodecomposition [in PCP] was observed, both in solution and in solid films, light probably is relatively unimportant in loss...from the environment." This implies that other processes are more significant than photodegradation for PCP loss. The authors did not discuss these processes, nor their reasoning for the statement.

Though it is difficult to draw any conclusions from these experiments, photolysis of PCP in water suggests that this may occur in the atmosphere also. Because of normal atmospheric humidity, there is water vapor in the atmosphere, and PCP is likely to adsorb onto aerosols due to its low vapor pressure and high partition coefficient. These particles could serve as nuclei for water droplet formation or could be washed out by rain. PCP has been found as a constituent of rainfall (Bevenue et al., 1972). Photolysis of sorbed PCP in the presence of oxygen also occurs by UV light. Sunlight intensity in the atmosphere will be much greater than in water, leading to a higher photolysis rate. Also, the results from the first experiment (Gäb et al., 1975) indicate that the presence of oxygen and UV light at wavelengths found in the atmosphere are sufficient for PCP degradation in either the sorbed or film state, but no light intensity was given. Results of both experiments indicate the formation of degradation products similar to those found in water photolysis.

On the basis of these considerations, the conservative assumption that PCP photolyzes in the atmosphere has been adopted. In water, a characteristic time can be taken to be approximately a week, but since no strong argument can be made for photolysis lifetimes in air in the range of hours or days, other removal processes have to be considered in order to determine a characteristic lifetime of PCP in air.

ii. Atmospheric Free Radical Oxidation

Though no direct information was found regarding the susceptibility of pentachlorophenol to free radical oxidation in the atmosphere, this is not expected to be an important fate process. Data from Darnall et al. (1976), the State of California Air Resources Board (1976) and Hendry and Kenley (1979) indicate that related compounds such as benzene, chlorobenzenes, nitrobenzenes, and phenol have low reactivity with respect to the OH radical in the air. The non-reactive rating is uncertain but can be reasonably estimated. The rate constant for OH radical oxidation of PCP is expected to be $<10^{-7}$/sec, based on the reactivity of these similar compounds ($<5 \times 10^{10}$ cm^3/mole-sec) and the global average OH concentration of 1.8×10^{-18} mole/cm^3. A similar analysis can be applied to ozone oxidation. The rate constant for benzene would be 4.5×10^{-11}/sec based on average ozone concentrations. If the similarity between PCP and benzene is valid, then ozone oxidation does not appear to be a significant atmospheric removal process.

iii. Rainout

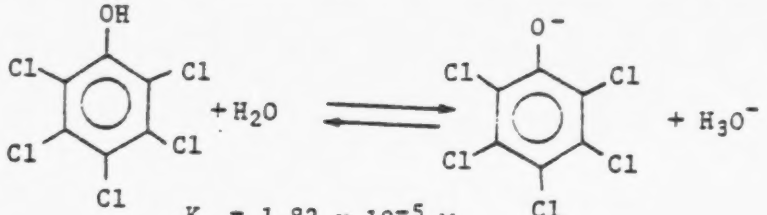
Rainout of PCP from the atmosphere is an environmental pathway that has to be considered. There are no data concerning the rate or frequency of the occurrence or the concentration of PCP in rainwater except for local situations (concentrations are calculated later in Section IV.E).

iv. Persistence

The residence time of pentachlorophenol in the atmosphere is dependent on its chemical reactivity, as well as on various atmospheric phenomena. Since PCP does not appear to be substantially degraded through photolysis or free radical oxidation, physical transport processes appear to the most important determinants of its atmospheric persistence. Time scales associated with major atmospheric processes have been estimated by Junge (1977) and Slinn (1978). For precipitation or nucleation scavenging of a generalized air-borne pollutant, they reported a time scale of one week and for the vertical mixing time of the troposphere, also one week. Based on these observations, one month was assumed as conservative estimate of the lifetime of PCP in the atmosphere.

c. Water

Pentachlorophenol is a moderately strong organic acid with a pKa of 4.74. PCP will therefore undergo a dissociation reaction with water, as shown in equation



$$K_a = 1.82 \times 10^{-5}\ M$$

From the dissociation constant, it can be calculated that PCP is extensively dissociated over the pH range (5-8) characteristic of natural waters. The ratio of pentachlorophenate anion to pentachlorophenol concentration is approximately 1.8 at pH 5, 18 at pH 6, 180 at pH 7 and 1800 at pH 8. The degree of dissociation affects the distribution of PCP in aquatic environments since the anion has a much greater water solubility than the parent acid (Table 21). The apparent values of other physical chemical parameters such as the Henry's law constant (Table 21) and octanol:water partition coefficient will also be pH dependent and these in turn will result in pH dependent behavior with regard to chemical reaction, adsorption onto biota or sediments and volatilization.

Unfortunately, the pH of the aquatic medium is not always stated in conjunction with reported experimental or monitoring data on PCP. This accounts for some apparent discrepancies in reported values. For example, the value of the octanol:water partition coefficient cited by Versar (1980) (Table 18) as 102,000 or log P = 5.01, probably refers to the relatively water insoluble PCP itself and would be appropriate for water with pH $\leq$ 3. The values given by Branson and Blau (1979) as 490 at 18°C (log P = 2.69) and 980 at 70°C (log P = 2.99) are probably "mixed" constants, reflecting the higher hydrophilicity of the (unknown) fraction of the PCP that was dissociated under the experimental conditions.

i. Hydrolysis

The covalent bond of a substituent attached to an aromatic ring is usually resistant to hydrolysis because of the high negative charge-density of the aromatic nucleus. For example, the synthesis of pentachlorophenolate anion from hexachlorobenzene requires treatment of the hexachlorobenzene with concentrated alkali at 130°C-200°C. It can be assumed that more extreme conditions would be necessary for further hydrolysis of the pentachlorophenolate anion. These are conditions not normally found in the natural environment. Consequently, hydrolysis is not an important mechanism for the removal of PCP from the environment (USEPA, 1979a).

ii. Photolysis

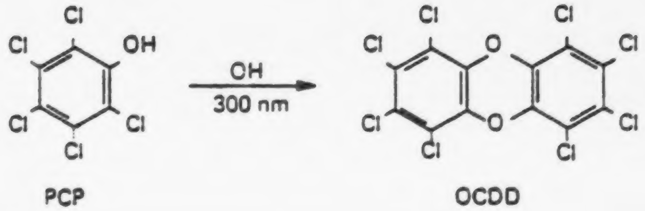
Pentachlorophenol in water is photodegraded by sunlight. PCP absorbs ultraviolet light in the solar spectrum, with maximum absorption occurring at a wavelength of 320 nm. In simulated sunlight, PCP at concentrations of 100 mg/l in a pH 7.3 borate-phosphate buffered solution was degraded totally in approximately 20 hours. Total degradation occurred in a similar solution exposed to natural sunlight for 5-7 days (Wong and Crosby, 1978). During one release event (Pierce and Victor, 1978), PCP appears to have been photodegraded into tetrachlorophenol before reaching the water into which it flowed. This occurred while PCP was in a solution with fuel oil or while in a holding pond.

Although it has been reported that sunlight will not degrade PCP in the absence of water or some other solvent (Arsenault, 1976), evidence exists to contradict this statement (Gab et al., 1975).

Several intermediate products will be formed as a result of PCP photodegradation (Wong and Crosby, 1978; Kaufman 1978a; Crosby and Wong, 1976). In naturally alkaline waters, it is possible that chlorinated dioxins could be formed photochemically if the PCP concentration were sufficiently high for bimolecular interaction. The production of dioxins would depend then on the photolysis rate and concentration. The concentration though is usually too low for dioxin formation (Crosby and Wong, 1976) (see Figure 7).

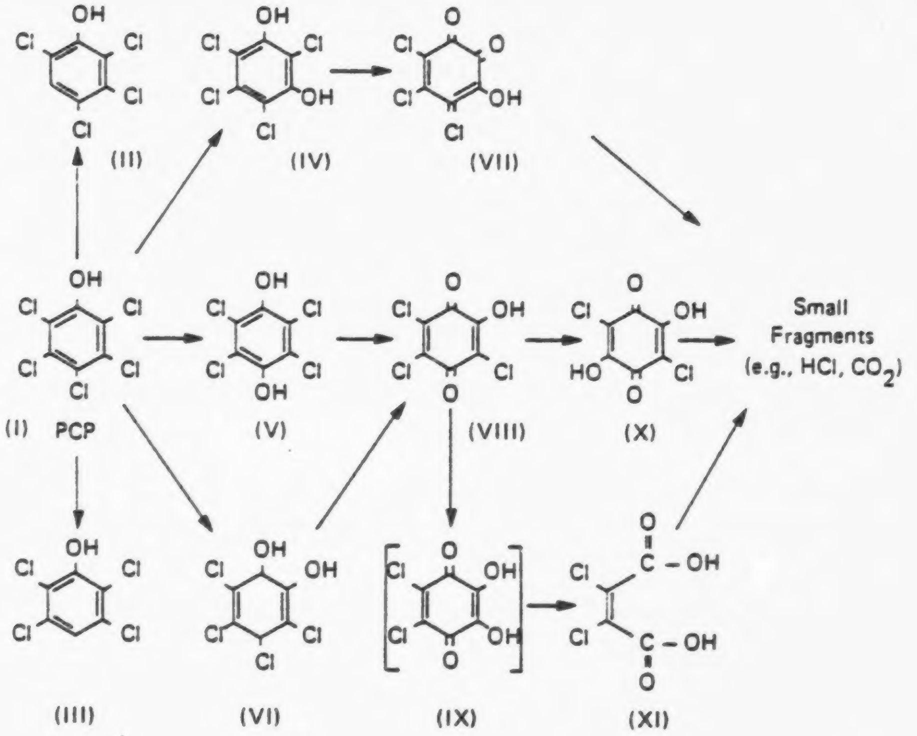
Test compounds irradiated in simulated and natural sunlight generate octachlorodibenzodioxins (OCDD) and hexachlorodibenzodioxins (HCDD), but no di-, tri-, or tetrachlorodibenzodioxins (Crosby and Wong, 1976). These latter compounds are also unstable in ultraviolet light so may degrade too rapidly for detection even if they are formed. The presence of HCDD indicates that the more stable OCDD is being photodegraded. Pure PCP in an aqueous solution of NaOH at a PCP (pentachlorophenate anion) concentration of 10 mg/l irradiated in simulated sunlight for 16 hours generated 26 mg/l of OCDD and 5 mg/l of HCDD. HCDD and OCDD are also trace contaminants in technical grade PCP (Arsenault, 1976), and so may be present before they are formed as photodegradation products.

Degrading PCP also forms other compounds, lower chlorinated phenols, tetrachlorodiols, and nonaromatics, as it degrades into final products such as CO_2, HCl, H and chloride ions. A pathway for PCP degradation and a list of products corresponding to these compunds are shown in Figure 8 (Wong and Crosby, 1978); Kaufman, 1978a). PCP-OCH_3 had also been found in the aquatic environment as a product of PCP degradation and it is speculated that this compound forms in the sediment in the water (Pierce and Victor, 1978).


OH
300 nm
PCP
OCDD

Source: Crosby and Wong, 1976.

FIGURE 7 DIOXIN FORMATION FROM PCP PHOTODEGRADATION


(I) PCP
(II)
(III)
(IV)
(V)
(VI)
(VII)
(VIII)
(IX)
(X)
(XI)
Small Fragments (e.g., HCl, CO$_2$)

I PCP
II 2,3,4,6-tetrachlorophenol
III 2,3,5,6-tetrachlorophenol
IV tetrachlororesorcinol
V tetrachlorohydroquinone
VI tetrachlorocatechol
VII trichlorobenzoquinone
VIII 3,5,6 trichloro-4-hydroxybenzoquinone
IX 2,3-dichloro-5, 6-dihydroxybenzoquinone
X 2,5-dichloro-3, 6-dihydroxybenzoquinone (chloranilic acid)
XI 2,3-dichloromaleic acid

Sources: Wong and Crosby, 1978.
Kaufman, 1978a.

FIGURE 8 PROPOSED PHOTOLYSIS PATHWAY FOR PCP

Sodium pentachlorophenate (NaPCP), the sodium salt of PCP, is quickly degraded in the presence of ultraviolet light and daylight. Degradation is almost complete in 16 hours with a sun simulating lamp (Stehl et al., 1971) (see Table 20). NaPCP has been shown to degrade rapidly in shallow streams exposed to sunlight. NaPCP is sensitive to wavelengths in the solar spectrum between 290 nm and 330 nm. The degradation process appears to follow first-order kinetics with a velocity constant $k=3.4 \times 10^{-4}$/sec (Hiatt et al., 1960; Monakata and Kuwahara, 1978) at $I=0.04 W/cm^2$ between 290 and 330 nm. The degradation rate equation is

$$\frac{dc}{dt} = \frac{kI_o t \ (1-e^{-\alpha L})}{\alpha L} \quad c$$

where k = a velocity constant, sec^{-1},

I_o = light intensity, w/cm,

α = absorption coefficient,

L = water depth,

t = time, and,

c = concentration.

Hiatt et al. (1960) reported $I_o=0.56 w/cm^2$ between 290 nm and 330 nm at Cleveland at noon in midsummer. This value is apparently high by a factor of five or more, since solar intensity outside the atmosphere is only 0.135 w/cm^2. At sea level, sun at Zenith, incident light intensity over the whole spectrum is 0.106 w/cm^2 (Loferski, 1956). Consequently, this equation may not be applicable to environmental conditions. The 0.04 w/cm^2 value seems more realistic.

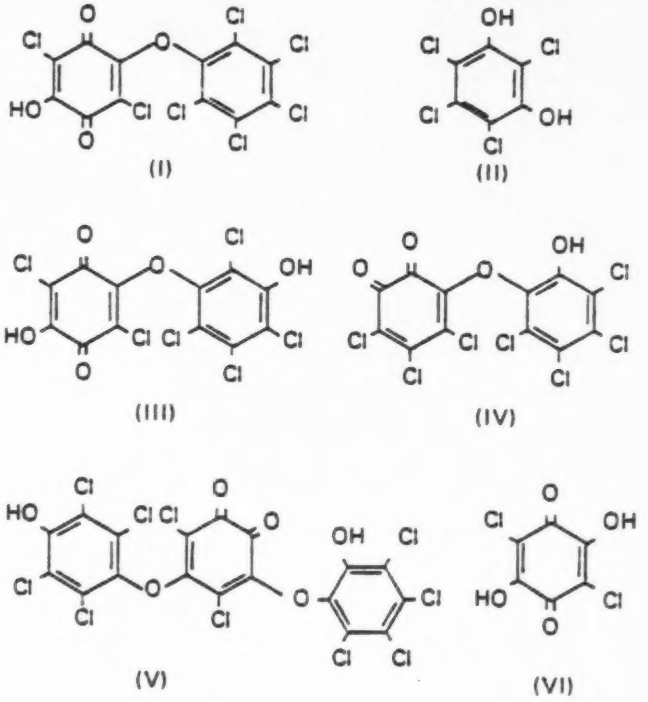
TABLE 20. STABILITY OF NaPCP IN SUNLIGHT AND ARTIFICIAL LIGHT IN pH 8 PHOSPHATE BUFFER

Light Source	Exposure Time	% Degradation
Control	0	0
Fluorescent Black Light	16 hr	7.8
Mineral Light	16 hr	18.7
G.E.R.S. Sunlamp	16 hr	93.9
Daylight	12-16 hr [1]	45.6

[1] Calculated from a total of 5 days, of which 3.5 days were rainy and 1.5 days (12-16 hr) had sunlight.

Source: Stehl et al., 1971.

The reported products of NaPCP photodegradation are shown in Figure 9.



I 2,5 dichloro-3-hydroxy-6-pentachloropehnoxy-p-benzoquinone
II 2,4,5,6-tetrachlororesorcinol
III 2,5 dichloro-3-hydroxy-6-(2',4',5',6'-tetrachloro-3'- hydroxyphenoxy)-p-benzoquinone
IV 3,4,5 trichloro-6-(2',3',4',5'-tetrachloro-6'-hydroxyphenoxy)-0-benzoquinone
V a 3-ring chloro, hydroxy compound
VI chloranilic acid

FIGURE 9 NaPCP PHOTODEGRADATION PRODUCTS

Conclusion: Pentachlorophenol has been shown to photolyze in water under conditions found in the environment. Degradation products formed may include octachlorodibenzodioxins and hexachlorodibenzodioxins, though these may also be present as contaminants in technical grade PCP. Final degradation products are CO_2, HCl, H and chloride ions. The sodium salt of PCP also photodegrades in water, and degradation products are similar to those of PCP. No degradation rate constants applicable to environmental conditions were found; however, a week seems to be a reasonable lifetime to expect based on reported experiments.

iii. Oxidation

Although there is no specific information about the oxidation of PCP, it can be assumed that this chemical behaves in the same manner as other highly chlorinated organic compounds. Chemicals within this class are usually resistent to oxidation even at high temperatures not commonly found in the natural environment (Morrison and Boyd, 1973). For this reason, oxidation is assumed not to be an important chemical process in the degradation of PCP under ambient conditions (USEPA, 1979a).

iv. Volatilization

Pentachlorophenol is not expected to volatilize from water at an appreciable rate. The vapor pressure of PCP at 20°C is 1.1×10^{-4} torr (1.4×10^{-7} atm) and its solubility at pH6 is 75 mg/1 (.28 mole/m^3) (see Table 21). The Henry's Law coefficient is estimated to be (MacKay, 1978; MacKay and Yuen, 1979). $H = (\text{Vapor Pressure})/(\text{Solubility}) = 5.2 \times 10^{-7}$ atm-m^3/mole. At higher pH values, H is lower, as shown in Table 21. If the value of H is less than about 3×10^{-7} atm-m^3/mole, the substance is less volatile than water and its concentration will increase as water evaporates. Humidity in the air will lower the volatilization rate of water somewhat so the lower limit is set at about 10^{-7}. Therefore, a substance in the range of $H=10^{-7}$ to 10^{-6} atm-m /mole can be considered involatile, or may volatilize very slowly at a rate dependent on H.

For these reasons, volatilization from water will not be considered as an intermedia transfer process. This does not preclude PCP input into air during spraying or mechanical agitation, as might be the case in use in cooling towers or aeration/evaporation ponds.

TABLE 21. SOLUBILITY IN WATER AND HENRY'S LAW CONSTANT OF PENTACHLOROPHENOL AT DIFFERENT pH VALUES

pH	% Dissociated	Water Solubility (mg/l)	Water Solubility /mole/m^3	Apparent H,atm-m^3/mole
pH 3	1.8	14	0.053	2.7×10^{-6}
pH 5	64	34	0.13	1.1×10^{-6}
pH 6	95	75	0.28	5.2×10^{-7}
pH 7	99.5	1,950	7.32	2.0×10^{-8}
pH 8	99.95	19,300	72.5	2.0×10^{-9}

Note: Vapor pressure = 1.1×10^{-4} torr (1.4×10^{-7} atm) for pentachlorophenol. Increased solubility as a function of (pH) is due to increased fraction of pentachlorophenate anion.

Source: Branson and Blau, 1979.

v. Biodegradation and Adsorption

Very little is known about the biodegradation of PCP in natural waters. Most of the PCP emitted to a body of water is though to be absorbed by the suspended particulates, the sediment, and the leaf litter.

In one study in which the background concentration of PCP in a control pond was measured, the concentration of PCP was shown to be 0.5 µg/l in water and suspended particulates, 5 µg/kg in the sediments and 50 µg/kg in fish and leaf litter (Pierce *et al.*, 1977). This division of PCP among the suspended particulates, the sediment, and the leaf litter is typical of the way PCP partitions in the aquatic environment. Little will go to the suspended particulates unless there is an unusually large suspended particulate load, more will go to the sediment, and most will be absorbed by the leaf litter.

The same study investigated the concentration distributions in an aquatic ecosystem after a sudden large spill of PCP into its waters. Concentrations were measured at six sites starting approximately 2 months after the spill and continuing every 2 months for a year and a half. Except at the first sampling time, the concentration of PCP in the sediment was far more than the concentration found in the water. At one site, the concentrations in the two media differed by a factor of a hundred (see Table 22). The low level of PCP that continued to be found in the water was attributed to the continuous inflow of PCP from the contaminated watershed area connecting the source of PCP and the lake under study.

Yet still further evidence that PCP has a short life time in the water was found 2 months after the spill. The fish kill immediately after the spill was described as extensive to total Air and Water control

TABLE 22. PCP CONCENTRATIONS IN WATER AND SEDIMENTS

Sample Date		Sample Site 1	2	3	4	5	6
2/27/75	Water[1]	9	11	6	9	19	8
	Sediment[2]	800	<5	22	26	119	2
4/24/75	Water	11	6	8	8	9	15
	Sediment	1,160	36	21	98	180	34
6/28/75	Water	13	8	2	3	2	82
	Sediment	1,300	5	5	583	860	92
8/5/75	Water	-	-	3	2	3	1
	Sediment	-	-	10	205	207	42
10/11/75	Water	10	5	<1	<1	<1	<1
	Sediment	927	471	48	91	56	24
12/6/75	Water	76	25	1	2	2	-
	Sediment	163	900	11	97	97	-
2/7/76	Water	29	-	15	10	10	26
	Sediment	100	-	313	10	84	81
5/3/76	Water	18	-	2	-	2	2
	Sediment	96	-	20	-	21	7

[1] Reported as μg PCP/liter water.

[2] Reported as μg PCP/kg air-dried sediment.

Source: Pierce *et al.*, 1977.

Commission, Mississippi, 1975). However, within 2 months, a repopulation of the fish in the lake demonstrated that the concentration of PCP in water had drastically and rapidly declined. This could be attributed to acclimitization, though levels in the lake were also low, as Table 22 shows.

Throughout this investigation, the concentration of PCP in the leaf litter remained very high, even considerably higher than the concentration of PCP in the sediment. In a separate study (Pierce et al., 1974) it was shown that PCP-contaminated leaf litter released 10% of the PCP to water after a 24-hour equilibrium period. These results suggest that leaf litter and sediments once contaminated with PCP can serve as a continual source of pollution to the aquatic environment.

vi. Water Treatment

There are some discrepancies in the available data concerning the effectiveness of wastewater treatment on the removal of PCP from process wastes. Vela and Kasper (1973) reported an unsuccessful search for microorganisms capable of degrading PCP in industrial waters while Kirsch and Etzel (1973) reported some PCP degradation in laboratory cultures of mixed bacterial populations. Other investigators (Kincannon et al., 1967, Reiner et al. 1978, and Vela and Rainer, 1976) have concluded from their studies that PCP is removed in wastewater treatment.

In other studies, the effectiveness of activated sludge on the removal of pentachlorophenol was shown to be dependent upon the type of PCP, whether a pure sample or mixed samples of commercially available PCP preparations. Kirsch and Etzel (1973) examined the feasibility of biological treatment of PCP-containing wastes by using aerated continuous flow systems and a contrived, soluble, PCP-supplemented waste. The average degradation for PCP for 19 days of operation was 99.4%. On the 19th day, the high-purity PCP was replaced by a blend of composite of five commercial PCP solutions and a very noticeable decrease of the biodegradability of PCP was observed. Kirsch (undated) found the same result when commercial grade pentachlorophenate replaced "high grade" pentachlorophenate in experimental waste treatment.

In an investigation of biological treatment of PCP waste in a sewage treatment plant, Arsenault examined mixtures of PCP in aeration lagoon influent under aeration conditions (Arsenault, 1976). In two separate experiments, the concentration of pentachlorophenol dropped from 39.5 mg/l to 0.5 mg/l in 3 days and from 81.0 mg/l to 0.6 mg/l in 30 hours. These results show that secondary treatment is effective in reducing the concentration of pentachlorophenol.

An on-site testing of the efficacy of pentachlorophenol removal by water treatment was conducted in Corvallis, Eugene, and Salem, Oregon (Buhler et al., 1972). The level of PCP in 24-hour composite samples of sewage influent was collected simultaneously from these three cities and was shown by gas chromatography to be 1-5 μg/l. Composite effluent values from the same sewage treatment plant showed 1-4 μg/l PCP, representing a 4-28% removal. Examination of water from Willamette River just upstream from Corvallis, Oregon, showed a considerably smaller concentration, only 0.10-0.70 μg/l. However, conventional processing of raw Willamette River water at the nearby drinking water treatment plant showed a removal of 60% of the PCP originally present in the water. At the Corvallis, Oregon, sewage treatment plant, water samples were taken at each stage of the treatment plant to help determine the most efficient stage for the removal of chlorophenol. As Table 23 shows, most of the PCP was removed by the trickling filter, a finding suggesting that PCP was subjected to substantial biodegradation.

d. Soil

i. Adsorption and Leaching

Adsorption: PCP is adsorbed most strongly onto soils that are strongly acidic, regardless of the species of clay mineral or the organic matter content (Choi et al., 1974). Choi et al. (1974) studied 13 soils of different clay and organic matter makeup, with concentrations of PCP varying from 12.5 mg/kg to 500 mg/kg. The soils were divided into three groups according to the pH: 4.6 to 5.1, 5.6 to 6.3, and 6.7 to 6.8 for groups one, two and three, respectively. For group one, the adsorption relationship was linear over the concentration range used. For the middle group the relationship was curvilinear, while group three showed practically no adsorption. Since the initial PCP solution was alkaline, PCP was almost completed dissociated. When this solution was added to the group one acidic soil, PCP anions underwent protonation. At concentrations greater than the solubility of PCP, then precipitation occurred (Choi et al., 1974).

For the second group in which a moderate amount of adsorption occurred, the pH of the soil-PCP system was 5.6 or greater. Therefore, PCP was completely dissolved and mostly dissociated. However, the pH of the clay surface was lower than the pH of the external solution (liquid phase) in the case of partly neutralized soils. Consequently, it was possible for PCP molecules to exist at the clay surface even when the pH of the liquid phase was high enough for complete dissociation. PCP adsorbed on the soil would be PCP molecules and/or anions (Choi et al., 1974).

For group three where the pH is 2 units or more greater than the pK_a of PCP, there was no adsorption or precipitation. Apparently, these soils do not adsorb the PCP anions.

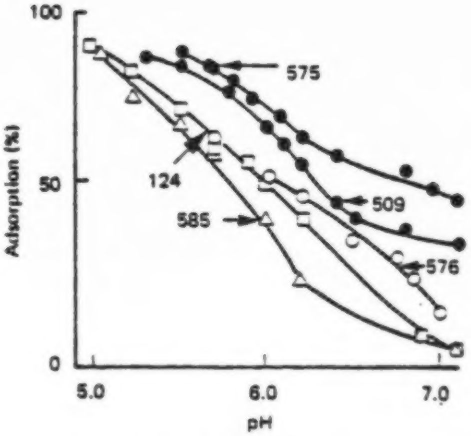
PCP adsorption onto soils is apparently a function of the pH of the soil. Figure 10 graphically illustrates this relationship. As pH increases, the percent of PCP adsorbed decreases. However, it is interesting to note the difference in adsorptivity among the soil types at a given pH value. The magnitude of adsorption appears to be related to the nature of soil colloids. The decreasing order to adsorption is humus-rich

TABLE 23. CONCENTRATION AND PERCENT REMOVAL OF HEXACHLOROPHENE AND PENTACHLOROPHENOL AT VARIOUS STAGES WITHIN THE CORVALLIS, OREGON, SEWAGE TREATMENT PLANT[1]

Stage Sampled in Plant	Time Sampled[2]	Concentration[3] (µg/l)		Percent Removal	
		HCP	PCP	HCP	PCP
After grit chamber (influent)	9:00	22.2	0.91	...	...
After primary clarifier	10:00	21.6	1.10	2.7	0.0
After trickling filter	10:30	6.4	0.51	71.4	44.0
After chlorination	11:00	6.8	0.49	69.4	46.2
Out to river (effluent)	13:00	7.3	0.33	67.1	63.7

[1] Samples collected on September 24, 1969.
[2] Sampled at the times indicated to compensate for flow times between the various stages of operation
[3] Mean of duplicate extractions and analyses.

Source: Buhler *et al.*, 1972.



Note: Numbers represent different soil types.

Source: Choi, et al., 1974.

FIGURE 10 RELATION OF THE APPARENT ADSORPTION TO THE pH OF THE SUPERNATANT LIQUID (Initial concentration: 100 ppm)

allophanic (Nos. 575 and 509), allophanic (No. 576), montmorillonitic (No. 124), and halloysitic soil (No. 585). Humus seems to adsorb more PCP than inorganic clays, and allophanic clay does so compared with crystalline clay (Choi et al. 1974).

Much research has been done concerning adsorption of PCP on the organic fraction of soil. An increase in organic matter increases the adsorption of PCP in soils. (A more detailed review of this subject is given in the Section IV.C.d.iv. below).

Leaching: Leaching does not appear to be a significant mode of transport for PCP due to the chemical's high affinity for binding to organic matter in soil and its low solubility in water. Any leaching that does occur is by the movement of the oil-based solution. In addition, some leaching may take place in alkaline or in inorganic clay soils with a low organic fraction; however, no specific information on this subject was available.

In one test, leachates were captured from soil that was composted with PCP-containing sludges from a wood preservation process at concentrations of 100, 200, and 300 mg/l of PCP (Arsenault, 1976). Concentrations in the leachates were 0.19, 0.067 and 0.108 mg/l, respectively, less than 0.1% of the original amount or concentration of PCP. Consequently, it was concluded that there is not significant migration of PCP through the soil.

ii. Photolysis

No information was found concerning the photolysis of PCP in soil. It is assumed, however, that this would be only a minor degradation process since only small amounts would be exposed on the soil surface. Photolysis of PCP in soil will, therefore, not be considered further as a degradation process.

iii. Volatilization

PCP sorbs strongly onto soils and sediments, especially those with high organic matter content. Therefore, loss from soil by volatilization is not expected to be a major transfer pathway. In an experimental setup measuring photodegradation in soil, losses by volatilization accounted for only 0.5% or less of the PCP added to the soil (Kaufman, 1978).

iv. Biodegradation

The rate of biodegradation of PCP is largely dependent upon the temperature, aeration, moisture, and organic content of the soil; somewhat dependent on the cation exchange capacity and soil pH; and not dependent upon the soil texture, clay content, or degree of base saturation. Kuwatsuka and Igarishi (1975) reported a weak correlation of PCP biodegradation rates with free iron content and phosphate adsorption coefficient, but little correlation with available phosphorus content. Their conclusion concerning free iron content can be contrasted with Kaufman's (1978b) finding that the rate of degradation of PCP is not influenced by free iron oxides.

The effect of flooding and anaerobic conditions on PCP degradation in rice paddies and upland adjacent fields was investigated under field conditions (Kuwatsuka and Igarishi, 1975). It was shown that the half-life for PCP under flooded conditions ranged from 10 days to 70 days, while under upland conditions the range was 20 to 120 days. The time required to degrade 90% of the PCP present was 80 days or more under flooded conditions and 50 days or more under dry conditions (see Figure 11 and Table 24).

Temperature also plays a key role in the rate of PCP degradation in soil. At higher temperatures, PCP disappeared more quickly under anaerobic (flooded) conditions than it did under aerobic conditions.

The most influential factor in the degradation of PCP is the amount of organic material present in the soil. In a laboratory study, Kuwatsuka (1972) found that the half-life of PCP at an initial concentration of 100 mg/kg at 30°C was 10 to 40 days under flooded conditions in arable soil, whereas almost 100% was present in soils with low organic matter content after 2 months (see Figure 12). Kuwatsuka and Igarishi (1975) showed that a forest subsoil with only trace organic matter retained almost 100% of the initial PCP concentration after 50 days under both water conditions. Young and Carroll (1951) also observed that PCP decreased more rapidly in soils containing a higher organic matter content. Results found by Watanabe (1973a) and Ide et al. (1972) revealed that PCP disappeared quickly in mature paddy fields, but very slowly in a newly reclaimed field and in soil collected from an immature field. No reasons for this behavior were given, but it may be due to the presence of adapted populations.

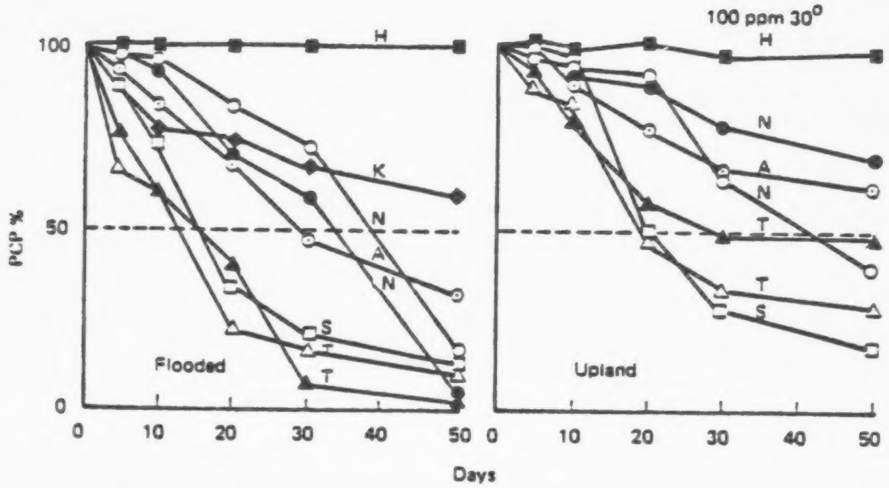
The same investigators found that additional applications of PCP to the paddy fields resulted in accelerated degradation and that no PCP degradation occurred in submerged soils that had been sterilized. These results indicate that PCP degrades more quickly in soils with high organic matter and that the degradation process is related to the microbial activity of the soil. A final study by Kaufman (1978b) showed a more rapid degradation of PCP in paddy soil under flooded conditions than in previously aerobic soils placed under anaerobic conditions, thus showing that the rate of degradation is dependent on an established anaerobic population.

Appendix B presents more detailed information on biodegradation products and pathways for pentachlorophenol.

e. Treated Wood

i. Photolysis

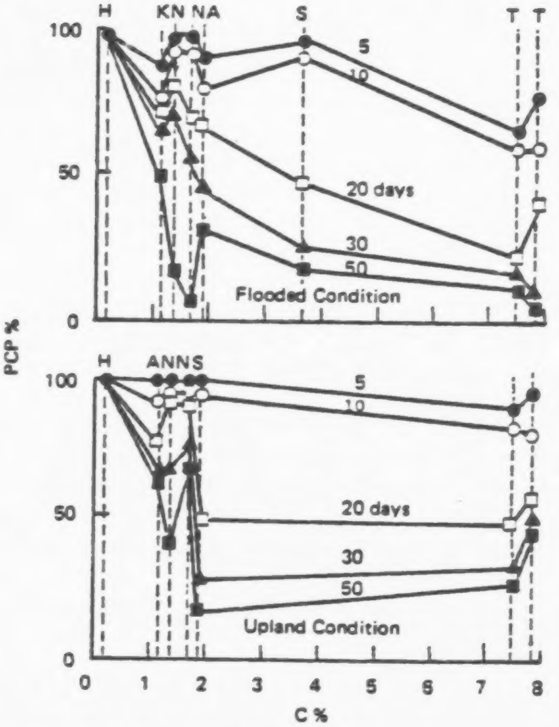
Pentachlorophenol on the surfaces of treated wood products exposed exposed to sunlight is photolyzed (USEPA, 1978c, Lamparski et al., 1980). PCP concentration on wood poles treated with PCP/oil solution on the sun-exposed side of the wood and the northern side of the wood



Note: Letters indicate different soil types.

Source: Kuwatsuku (1972).

FIGURE 11 PCP DEGRADATION IN VARIOUS SOILS UNDER FLOODED AND UPLAND CONDITIONS



Source: Kuwatsuku (1972)

FIGURE 12 RELATION BETWEEN DEGRADATION RATE OF PCP AND ORGANIC MATTER CONTENT IN VARIOUS SOILS FOR DIFFERENT INCUBATION PERIODS (days)

TABLE 24. CORRELATION COEFFICIENTS BETWEEN DEGRADATION RATE OF PCP AND PROPERTIES OF SOILS[1]

Incubation period (days)	Flooded Conditions				Upland Conditions			
	5	10	20	30	5	10	20	30
Soil Property								
Total Carbon Content	***0.839	***0.815	**0.625	***0.827	**0.817	**0.814	*0.539	0.383
Clay Content	0.182	0.457	0.352	0.432	0.046	0.264	0.194	0.152
C.E.C.	***0.831	*0.574	*0.599	*0.525	**0.926	**0.763	0.368	0.333
Available Phosphorus Content	0.392	0.365	*0.499	*0.589	*0.675	*0.642	0.144	0.198
Free Iron Content	**0.889	*0.633	***0.858	0.409	0.091	0.597	0.358	0.387
pH (H O)	0.392	*0.630	0.420	0.432	0.181	0.539	0.444	0.153
Phosphate absorption Coefficient	***0.877	*0.730	***0.908	***0.922	***0.937	**0.821	0.368	0.253

[1] *** ** * indicate significance levels of 0.1, 1, and 5%, respectively

[2] Data on Tochigi soils under flooded and upland conditions were not included.

[3] Degradation rate = $\frac{\text{Applied amount + residual amount}}{\text{Applied amount}} \times \frac{1}{\text{Incubation Period}}$

Source: Ide et al., 1973.

(shaded side) differed by 2.4 ng/in^2. On the sunny side PCP was found on the surface at .7 ng/in^2 and on the shady side at 3.1 ng/in^2. On wood poles treated with a PCP/cella mixture, the differences were even greater. PCP was found on the south side on the surface at a concentration of 0.2 ng/in^2. On the north side the concentration of PCP was 2.8 ng/in^2. This is a difference of 1.74 ng/in^2 in three years, or .58 ng/in^2/yr. This figure probably does not represent the actual photolysis rate since initial conditions were not known and some portion of the difference observed may be attributable to other causes.

Experiments with PCP added to filter paper showed that exposure to ultraviolet light for 7 days degrades more than 90% of 7500 ng PCP initially applied (USEPA, 1978c). There is some evidence that hexachlorobenzene and certain dioxins may be degradation products of PCP photolysis (U.S. EPA 1980a).

Experiments were performed to determine the effects of sunlight on chlorinated dibenzo-p-dioxin formation from PCP in wood treated with different types of PCP (Lamparski et al., 1980). Yellow pine was treated with Dowicide EC-7 anti-microbial PCP, purified PCP, and technical PCP in two types of carrier solutions, methylene chloride and P-9 oil. Samples were exposed to natural sunlight and a GE Model RS sunlamp that approximates the natural sunlight. Results of the experiments showed that EC-7 behaved similarly to very pure PCP. An increase in octachlorodibenzo-p-dioxin (OCDD) concentration was noted in the solutions using methylene chloride,peaking after about 20 days and was due to photolysis. Hexa-CDD and hepta-CDD were also found, and temporal variation was similar to that of OCDD, and were apparently a photodegradation product of OCDD. Temporal behavior of OCDD formation in the PCP/P-9 oil mixture was similar to that of the PCP/MC mixture, though total OCDD concentrations were significantly less (by a factor of about 20) in the PCP/P-9 oil mixture. The higher content in the PCP/MC solution was attributed to photolytic reactions of the PCP with the methylene chloride. This did not occur in the PCP/P-9 oil solution. No information was reported concerning the concentration of PCP as a function of time.

In conclusion, PCP residing on wood surfaces exposed to light is likely to be photodegraded. It should be noted that the amount of PCP held in the surface layer of treated wood is small relative to the unavailable amount held in the body of the wood. Unless PCP solution continues to migrate out from underlying layers of wood, the PCP concentration on the wood surface will decrease due to photolysis (Arsenault, 1976).

ii. Volatilization

The vapor pressure of pentachlorophenol, 1.1-1.7 x 10^{-4} torr at 20°C and 3.1 x 10^{-3} torr at 50°C, suggests that PCP may be volatilized from wood surfaces (USEPA, 1978a). Walls in a room treated with a PCP wood preservative released the chemical into the air, with concentrations reaching 1 ng/m^3 air on the first day after treatment and 160 ng/m^3 air on the fourth day (Gebefugl et al., 1970). Since no information was given about the size of the room, treated surface/enclosed volume rates, or air exchange rates, it is not possible to compute the volatilization rate.

The EPA (1978c) reports that evaporation of PCP, with a vapor pressure of 1.6×10^{-4} torr, from a treated surface in which it is adsorbed yields a vapor density of 2.3×10^{-9} g/cm^3 in the immediate vicinity of the surface. This yields a calculated rate of evaporation of 1.7×10^{-10} g/cm^2/sec or 53.6 g/m^2/yr, though no method for this calculation was given. Due to the high adsorption forces for PCP on wood, PCP may be volatilized much more slowly. Therefore, these values should be considered an upper bound on the rate. The results of more recent investigations on this subject are expected to be available in the near future (U.S. EPA 1980a).

In wood treatment, the PCP concentration in the finished products is about 8 kg/m^3 (see calculation developed subsequently in IV-D-2-c for PCP atmospheric concentrations due to volatilization from treated wood products). So, in the top 1 mm of surface of distribution, the "concentration" is 8 kg/m^3 $\times 10^{-3}$m = 8 g PCP/m^2 surface. The upper limit value of the volatilization rate indicates that this PCP would be depleted in 0.15 yr or 1.8 months. One year is, therefore, expected to be a conservative estimate for the lifetime of PCP in wood surfaces.

iii. Leaching

No information was available that would indicate the propensity for PCP to leach out of treated wood into surrounding soil or water. Although PCP has a high affinity for organic matter, it may behave differently in wood, especially under saturated conditions. If its adsorption to wood is also pH dependent, alkaline conditions may cause its release from wood. These statements are speculative and experimental verification is required before conclusions regarding PCP's behavior in wood can be made.

f. Conclusions

Based on the results of the laboratory and field studies of pentachlorophenol in air, water, soil and treated wood described in Section IV-B-1., some generalizations can be made concerning the pollutant's behavior in the environment. These conclusions are based on limited data and are subject to the usual difficulties in extrapolating experimental results to natural systems.

The air compartment appears to receive a large amount of PCP discharge, however, the total mass entering the atmosphere cannot be accurately estimated at this time (see Section III). Pathways for entry into air include evaporation from aeration/evaporation ponds at wood treatment facilities used instead of direct discharge to water, evaporation and/or droplet release from cooling towers using PCP biocide, volatilization from treated wood products and burning of treated wood. Physical removal mechanisms, such as rainout, are important processes affecting PCP concentrations in the atmosphere. Photolysis appears to be an important transformation process based on PCP's behavior in water. Free radical oxidation does not appear to affect PCP. The atmospheric lifetime for PCP was estimated at one month and was assumed to be determined primarily by physical removal processes.

Surface water is also an important compartment to consider in PCP pathways analysis. Certain PCP users practice direct discharge and there is potential for direct entry from numerous non-point sources including from treated wood. In addition, transfer of PCP from other compartments such as air through rainout, and soil, throughout runoff and leaching, can occur. Monitoring data and fish kill incidents indicate the presence of PCP in water over the past ten years (see Sections IV-A and VI-B). Unfortunately, data are few and scattered, and do not indicate the national distribution of the pollutant in surface waters. At low concentrations in water, PCP appears to be quite readily degraded by microbial action (in water treatment facilities) and, in clear water, photolyzes rapidly. Extreme concentrations or turbid conditions could inhibit these two processes. Sorption to sediments and larger pieces of organic matter is also likely to occur. Hydrolysis, oxidation and volatilization do not appear to affect concentrations in water significantly. The lifetime of PCP in natural waters in which PCP fate processes take place was estimated at one week, in conditions optimizing photolysis. For systems where biodegradation is the primary removal mechanism, the lifetime will be longer and extremely variable, depending on the conditions. Once PCP is adsorbed onto sediment, there appears to be potential for re-release to water; however, the conditions that would trigger this transfer have not been investigated.

Pentachlorophenol enters the soil compartment as a result of its use as a garden herbicide, leaching from treated wood, rainout from the atmosphere, and spills around PCP-using industries. No monitoring data of PCP levels in soil were available to indicate typical concentrations. Due to its high adsorptivity, especially on organic matter and under acidic conditions, and based on the results of a laboratory study, PCP does not appear to be very mobile in soil. Biodegradation is a significant removal process under both aerobic and anaerobic conditions. Important variables affecting the degradation rate include temperature, moisture and the presence of organic matter, and less so, the soil cation exchange capacity and pH. Photolysis and volatilization do not appear to be important soil processes in relation to PCP concentrations. Transfer of PCP from soil to water may occur through runoff and physical transport of PCP-adsorbed surface soil.

Wood that has been treated with pentachlorophenol apparently loses, at a minimum, the amount of chemical residing in the wood's surface layer. Photolysis degrades PCP in products exposed to sunlight, although no rate information was available. In addition volatilization occurs at a fairly slow rate, estimated at 1.7×10^{-10} g/cm^2/sec. Based on this volatilization rate, a lifetime of one year for PCP residence in wood was estimated. The potential for removal of PCP from treated wood resulting from runoff from surfaces or leaching exists, however, information that would indicate the overall importance of this process was not available. Further investigation of this potentially significant pathway to water and soil is essential because of the large amount of PCP used as a wood preservative and the widespread national distribution of treated wood products.

2. Biological Pathways

a. Introduction

This section concerns the biological pathways for PCP, and particularly its uptake and metabolism in higher organisms. The topics discussed include potential for uptake, bioaccumulation, metabolism, and elimination of PCP in both aquatic and terrestrial organisms.

b. Freshwater Systems

The potential for bioaccumulation of PCP is indicated by the log of the octanol/water partition coefficient, log P = 5.01. This potential is confirmed by a number of laboratory studies, which have indicated that PCP is accumulated significantly by aquatic organisms, particularly those from higher trophic levels. Lu *et al*. (1978) examined the fate of radiolabeled PCP in two model ecosystems, one simulating a fresh water system and the other a terrestrial/aquatic system simulating a lake and its shoreline. For the first system, the results showed that the radiolabeled PCP and its metabolites were rapidly accumulated after 3 days in the food chain organisms, and most of the parent compound was stored intact. In the terrestrial/aquatic model with the same species, PCP was applied at a rate of approximately 1.0 kg/ha, and dispersed by the feeding activities of salt marsh caterpillars. In this system, however, bioaccumulation factors were considerably higher (by a factor of 3 to >100) than in the aquatic model. PCP was also metabolized to a greater extent (primarily by reductive dechlorination) in the terrestrial/aquatic system. No explanation was offered for the difference in results for the two models.

Other studies have confirmed the propensity of PCP to accumulate in the tissues of exposed organisms. Glickman *et al*. (1978) found that radiolabeled PCP was rapidly taken up by rainbow trout; residues accumulated to 16 mg/kg in the liver. High concentrations (250 mg/kg) of PCP were detected in the bile, primarily as the glucuronide conjugate. In another study, guppies (*Lebistes reticulatus*) were killed by 3 mg/l PCP in 16-18 hours. The bioaccumulation factor during this period was approximately 35 (Stark, 1969).

Kobayashi and Akitake (1975) exposed goldfish to 0.1, 0.2, and 0.4 mg/l of radiolabeled PCP. The respective bioaccumulation factors measured after death were 1,000, 570, and 278. In each case, the residues found in the dead specimens were approximately 100 mg/kg, a finding indicating a maximum tolerance level for this species. Even during exposure, PCP was excreted rapidly by the goldfish; accumulation occurred when the rate of absorption exceeded the rate of excretion.

In a study by Hallas (NDG), guppies accumulated PCP up to 0.1% of their weight before dying from toxic effects. Of the PCP residues, 99% were metabolized, leading the author to suggest that the metabolites, and not PCP *per se*, were toxic to fish.

PCP residues were found in three species of fish in a lake receiving effluents from a pulp mill (Rudling, 1970). Lake concentrations averaged about 3 µg/1, while PCP residues in perch, pike, and eel ranged between 0.15 and 3.0 mg/kg. In another field study, Pierce et al. (1977) examined the effects of a major PCP discharge to a man-made lake in Mississippi. After an initial fish kill (described as extensive to total), residues in small fish returned to background levels within 10 months. However, this work does not provide any information regarding possible biomagnification in the aquatic food chain, as no predator fish were examined for PCP residues.

Holmberg et al. (1972) exposed the anadromous yellow eel (Anguilla anguilla) to PCP in freshwater and seawater to compare uptake in the different media. The eels in freshwater were exposed for 4 days, and the test organisms in saltwater for 8 days, both at concentrations of 100 µg/1. The respective bioaccumulation factors were 90 and 300, although uptake was initially more rapid in freshwater. The authors attributed the difference in the factors to the unequal exposure periods, and possibly the higher pH in the seawater (8.1, as opposed to 7.1 in freshwater). (For a discussion of the influence of pH on PCP activity, see Section VI.A.)

The available data on PCP bioaccumulation in freshwater organisms are summarized in Table 25.

c. Marine Fish

In a study of PCP uptake by sheepshead minnows, Parrish et al. (1978) found that juveniles had substantially higher bioaccumulation factors than adults or egg/embryos, in addition to a more rapid rate of uptake. Schimmel et al. (1978) performed residue analysis on dead specimens of two fish and two shrimp species from 96-hour toxicity bioassays. A longer-term experiment was also conducted on the Eastern oyster, (a filter feeder and hence a better accumulator than most species) for 28 days. Equilibrium concentrations were attained in 4 days, but depuration to non-detectable levels was equally rapid, occurring in 4 days also. The data from these studies are summarized in Table 26.

d. Terrestrial Organisms

Lu et al. (1978) applied radiolabeled PCP to a model terrestrial ecosystem, and found that only 1% had entered into animals after 20 days, and 48% into the soil. Corn plants (Zea mays) accumulated PCP rapidly, and metabolized 84% of the uptake. The prairie vole (Michrotus ochregaster) at the top of the food chain, had consumed virtually all of the plant and animal material in the system after 5 days of exposure. Tissue residues accounted for 0.5% of the total PCP dose applied to the system, and ranged from 135 µg/kg in the uterus to 26 µg/kg in the skin.

In a field ecosystem study, Vermeer et al. (1974) examined uptake and effects of PCP and other pesticides on organisms in an 8000-ha rice field. Residues of PCP in birds, fish, and snails were measured after

TABLE 25. PCP BIOACCUMULATION IN FRESHWATER BIOTA

Species	PCP Concentration in media (µg/ℓ)	Bioaccum. Factor	Exposure Period	Reference
Gambusia affinis (aquatic system)	2.7	2.96	72 h	Lu *et al.* (1978)
Gambusia affinis (terr./aquatic sys.)	1.0 kg/ha	132	-	Lu *et al.* (1978)
Snail (*Physa sp.*) (aquatic system)	2.7	1.21	72 h	Lu *et al.* (1978)
Snail (*Physa sp.*) (terr./aquatic sys.)	1.0 kg/ha	21	-	Lu *et al.* (1978)
Daphnia magna (aquatic system)	2.7	1.65	72 h	Lu *et al.* (1978)
Daphnia magna (terr./aquatic sys.)	1.0 kg/ha	204	-	Lu et al. (1978)
Mosquito (*Culex pipiens*) larvae (aquatic system)	2.7	16	72 h	Lu *et al.* (1978)
Mosquito (*Culex pipiens*) larvae (terr./aquatic sys.)	1.0 kg/ha	21	-	Lu *et al.* (1978)
Alga (*Oedogonium cardiacum*) (aquatic system)	2.7	1.5	72 h	Lu *et al.* (1978)
Alga (*Oedogonium cardiacum*) (terr./aquatic sys.)	1.0 kg/ha	5	-	Lu *et al.* (1978)
Rainbow trout (*Salmo gairdneri*)	20	~0.35	24 h	Glickman *et al.* (1978)
Guppy (*Lebistes reticulatus*)	3,000	~35	18 h	Stark (1969)
Goldfish (*Carassius auratus*)	100	1,000	120 h	Kobayashi and Akitake (1975)
Goldfish (*Carassius auratus*)	200	570	20 h	Kobayashi and Akitake (1975)
Goldfish (*Carassius auratus*)	400	278	6 h	Kobayashi and Akitake (1975)
Snail	2,000	50	30 h	Weinbach and Nolan (1956)
Perch (*Perca fluviatilis*)	3	50	Indefinite	Rudling (1970)
Northern pike (*Esox lucius*)	3	67	Indefinite	Rudling (1970)
Yellow eel (*Anguilla anguilla*)	3	1,000	Indefinite	Rudling (1970)
Yellow eel (*Anguilla anguilla*) freshwater	100	90	4 d	Holmberg *et al.* (1972)
Yellow eel (*Anguilla anguilla*) seawater	100	300	8 d	Holmberg *et al.* (1972)

TABLE 26. PCP BIOACCUMULATION IN MARINE BIOTA

Species	PCP Concentration (µg/1)	Bioaccumulation Factor	Exposure Period	Reference
Sheepshead minnows (*Cyprinodon variegatus*)	18			Parrish *et al*. (1978)
juveniles	18	48	28 d	Parrish *et al*. (1978)
adults	18	27	151 d	Parrish *et al*. (1978)
eggs/embryos	88	26	138 d	Parrish *et al*. (1978)
Longnose killifish (*Fundulis similis*)	85	41	96 h	Schimmel *et al*. (1978)
Striped mullet (*Mugil cephalus*)	85	79	96 h	Schimmel *et al*. (1978)
Grass shrimp (*Palaemonetes pugio*)	76	3.0	96 h	Schimmel *et al*. (1978)
Brown shrimp (*Penaeus aztecus*)	49	0.45	96 h	Schimmel *et al*. (1978)
Eastern oyster (*Crassostrea virginica*)	2.5	78	28 d	Schimmel *et al*. (1978)
Eastern oyster (*Crassostrea virginica*)	25	41	28 d	Schimmel *et al*. (1978)

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applications of 4 kg/ha NaPCP, resulting in a concentration of 4 mg/l. Fish found dead after applications contained 31-59 mg/kg PCP, while fish collected alive from unsprayed ditches contained 2-13 mg/kg PCP. In dead snails (Pomacea sp.), residues of 37 mg/kg PCP were found. Birds in the area were found to contain PCP residues ranging from 0.04 mg/kg in the purple gallinule (Porphyruls martinica), which feeds on rice seeds, to 10.0 mg/kg in a common egret (Egretta alba), which feeds on insects, fish and amphibians. Fifty (out of 279) snail kites, which feed almost exclusively on Pomacea, were found dead after NaPCP spraying. Concentrations of PCP in their brains, livers and kidneys were 11, 46 and 20 mg/kg respectively.

In a study of PCP fate in cotton, Miller and Aboul-Ela (1969) found that the toxicant accumulated to concentrations of 2 mg/kg in cottonseed kernels of closed bolls. In kernels of open bolls on the same plants, however, PCP was not detected, indicating that it may be translocated from hulls to developing seeds.

Hilton et al. (1970) applied PCP to sugarcane in 6-mg doses per plant, and found that 84% was retained by the leaves. When the leaves abscised naturally, virtually all of the remaining PCP was lost from the plant. Roots exposed to PCP in solution absorbed 99% of the toxicant, while the remaining 1% was delivered to the stalk and suckers.

Rice seedlings in one experiment absorbed 3% of PCP applied to the soil at a concentration corresponding to agricultural application. Residues in the seedlings were determined to be 90% PCP, and most of the remainder unidentified conjugates (Hague et al., 1978).

e. Conclusions

PCP has a high propensity to accumulate in the tissues of both aquatic and terrestrial biota. Reported bioconcentration factors in fish range from 3 to 1,000, depending on the species, duration of exposure, aqueous PCP concentration, and environmental factors such as salinity and pH. Studies in which biochemical analyses were performed indicate that PCP is metabolized to a large extent in fish tissues. The metabolites are apparently toxic as well, which in some species are not tolerated above a certain level. There is some evidence that PCP biomagnifies in food chains, but no conclusive data on this aspect are available.

Little information is available on the characteristics of PCP uptake in terrestrial ecosystems. Sugarcane and corn plants have been observed to accumulate PCP strongly, although plants vary in their tendency to metabolize the toxicant. PCP may also concentrate selectively in certain tissues, such as the leaves or roots, depending on the type of exposure.

D. CONCENTRATION ESTIMATES

1. Introduction

This section presents estimations of PCP concentrations in environmental media based on simple calculational procedures using materials balance, fate , and in some cases, monitoring data as input. Only the air and water compartments were considered. Soil was excluded due to the complexities of the medium and lack of simple calculational models. This does not mean to imply that soil is not an important environmental compartment for PCP residues. There is a significant potential for release of the pollutant to soil through garden herbicide use and leaching from treated wood.

The air compartment is an important recipient of PCP discharge as discussed in Sections III and IV-B. Important sources to air include evaporation of wood treatment facility wastewater, volatilization from treated wood surfaces, cooling tower water evaporation, and open burning of PCP-treated wood. For an assumed steady-state input of PCP, estimations were made of air concentrations on a national scale resulting from each source category. In addition, the propensity for transfer of PCP from air to water (or soil) through rainout was calculated. Last of all, two local-scale PCP concentration estimations were calculated:

1) in a plume downwind from a cooling tower using PCP as a biocide,

2) in a plume downwind from a wastewater evaporation pond.

The water compartment is potentially a significant recipient of PCP release although the sources are numerous, widespread, and often undefined. Reports of PCP-caused fish kills (see Section VI-B) indicate PCP contamination of water at harmful concentrations. The most significant user of PCP, the wood preserving industry, does not commonly practice direct discharge to surface water. The amount of PCP associated with other users which do discharge is relatively small. The amount discharged to POTW's based on available data, is small and would be reduced 50% to 100% by treatment processes. An unknown amount of PCP held in storage lagoons has the potential for release through leakage or overflow. There is an additional potential for indirect entry into water from non-point sources through runoff or leaching from soil in herbicide-treated areas, leaching out of treated wood products and rainout from contaminated air.

Due to insufficient monitoring to characterize total PCP releases to water, no attempt was made to estimate concentrations in water on a national scale. Concentrations were, however, estimated on a local scale, making use of typical effluent concentrations and flow data associated with industries practicing direct discharge. The computerized EXAMS (Exposure Analysis Modelling System) model was implemented to simulate concentrations in water and sediment of six types of generalized aquatic systems. The model is discussed in greater detail in the following section.

2. Air Models

a. Assumptions Used in Air Models

Concentration calculations made in this section are based on three main assumptions:

(1) The atmospheric lifetime for PCP is one month. The time scale for removal of chemicals from air due to nucleation and/or precipitation scavenging is approximately one week (Junge, 1977; Slinn, 1978). PCP is probably photolyzed in air, but a time scale for this is not known. One month is, therefore, a conservative estimate.

(2) Ninety percent of the PCP from fugitive emissions is released into the air over an area one-half the size of the continental United States. This is based on production patterns of PCP-treated products and an assumed relationship between use of PCP-treated products and population. Eighty-two percent of wood treatment plants are located in the eastern half of the country (Section III). Ninety percent of the population occupies about one-half of the land area of the U.S., and PCP use is assumed to be directly related to population.

(3) The altitude at which PCP mixing occurs is assumed to be 1 km. This value characterizes the vertical extent of surface-based pollution on a time scale of days, and as such is a conservative assumption on the basis of the 1-3 month residence time.

These assumptions give a mixing volume of $4.7 \times 10^{15} m^3$ in which at any time one month of PCP release will reside. In view of the possible invalidity of any of these assumptions or others used on the modelling order of magnitude bounds will be placed on the estimated concentrations.

b. Atmospheric Concentrations Due to Wood Treatment Wastewater Evaporation

In a study of the effects of PCP on a stream, oil slicks on the water surface were found to contain up to 5450 mg/l of PCP, while the water contained up to 10.5 mg/l. On the basis of these two values, an oil/water partition coefficient of about 520 is indicated. Octanol/water partition coefficients for PCP are given as 490 at 18°C and 980 at 70°C (Branson and Blau, 1979). An average value of 550 will be used in the following calculations.

In the wood preserving industry approximately 150 l of water is used per m^3 of wood treated (USEPA, 1979a). The wastewater effluent contains approximately 1 gm/l of oil and grease and 20 mg/l of PCP.

(USEPA, 1979a). From these values it can be estimated that there is approximately 150 g of oil and grease and 3 g PCP in the effluent stream per m^3 of wood treated. Before disposing of wastewater, most plants practice oil/water separation to remove the oil and grease, which is then recycled or disposed. The remaining water is sent to either a cooling tower or a holding pond equipped with aerators and sprayers where vaporization takes place.

Though some plants do discharge treated water to POTW's or streams, most practice some treatment through the methods described. As the water evaporates from the cooling towers or ponds, the PCP contained in the water is also assumed to evaporate.

Using values given above, and computing the partitioning of the PCP between the process water and the oil and grease, about 2 g PCP/m^3 treated wood remains in the process water at a concentration of 0.013 g/l (13 mg/l), and 1 g PCP/m^3 wood is removed along with the oil and grease. Production of PCP-treated wood in the main categories is about 1.8×10^6 m^3/yr, giving 3.6×10^3 kg/yr of PCP input to air via wastewater evaporation. In a similar analysis, Branson and Blau (1979) assumed 200 l/m^3 for process water use and the effluent water contained 28 mg/l of PCP, or 5.6 g/m^3 (4.2 g/m^3 if the previous estimate of 150 l/m^3 is used), a concentration similar in magnitude to the 2 g PCP/m^3 wood derived above. An inconsistency in the reported analysis (Branson and Blau, 1979) appears though, since it states that 0.18% of the total amount of PCP used ends up in the treated water. The values of 28 mg/l PCP in treated water, 200 l/m^3 water use, and 8 kg PCP/m^3 wood indicate only a 0.07% loss. A loss of 0.18% of the total amounts to 3×10^4 kg/yr while the 0.07% figure indicates 1.2×10^4 kg/yr. Nevertheless, these loss estimates are narrowly within the same order of magnitude.

Eighty two percent of the wood treatment plants are located in the eastern half of the country, as shown previously in Figure 3. Figure 4 shows that a corresponding amount of PCP is used and presumably lost in the regions containing the preponderance of these plants.

Eighty-two percent of the range estimating PCP atmospheric loading due to wastewater evaporation (3.6×10^3 - 3.0×10^4 kg) amounts to 3.0×10^3 to 2.4×10^4 kg PCP lost annually in the eastern region alone. Based on the dilution volume and lifetime in the atmosphere for PCP assumed, 250-2000 kg of PCP is dispersed in an atmospheric volume of 4.7×10^{15} m^3. The resulting PCP concentrations range from 5.2 to 43 $\times 10^{-11}$ g/m^3 or 0.05 to 0.40 ng/m^3. An order of magnitude of error either way in these estimates gives a range of 0.005 ng/m^3 to 4.3 ng/m^3.

c. Atmospheric Concentrations Due to Volatilization From PCP-Treated Wood Products

Treated wood products account for approximately 80% to 90% of PCP use in the United States (see Materials Balance). The 1976 production levels of PCP-treated wood products are shown in Table 27. Sizes assumed for pieces in each of the categories of wood products are shown in Table 28. The surface area and volume from which PCP is assumed to volatilize is also shown. The PCP is assumed to be distributed evenly through the wood, all of the PCP is assumed to volatilize from only the top 1 mm of surface, and no PCP is assumed to be degraded photolytically or metabolically. This is a conservative assumption since PCP probably escapes via other routes or is degraded to some extent.

TABLE 27. USAGE DATA FOR PENTACHLOROPHENOL BY WOOD PRODUCT

Product Category	Amount Treated (m^3)	Total PCP Used (Kg) (10^6kg)	% Total	Rate (kg/m^3)
Wood Poles	1,034,410	10.8	65%	4.9-12.7
Lumber, timber, plywood	392,890	2.6	15.6%	4.9-8.1
Fence posts	257,600	1.8	10.6%	4.9-8.1
Crossarms	128,600	0.91	5.5%	4.9-8.1
Pilings	10,420	0.09	0.5%	4.9-13.8
Cross ties	540	0.05	0.2%	5.6-8.1
Millwork	283,200	0.45	2.7%	---
	TOTAL	16.6		

Source: U.S. EPA (1980b).

Four product types--poles, lumber, fenceposts and crossarms--account for most of the PCP use as a wood preservative.

The total volume of treated wood from which PCP is assumed to volatilize per year is found to be 3.6 x 10^4 m^3 as shown in Table 28. The concentration in wood is used to determine the total amount of PCP volatilized (see Table 29). The upper limit is used. The total amount of material volatilizing each year can then be estimated as 3.44 x 10^5kg, or roughly 2% of the total amount of preservative applied. Based on the atmospheric lifetime assumptions of one month adopted previously, the total amount of preservative in the air at any one time is one twelfth this amount, or ~2.9 x 10^4kg.

TABLE 28. DIMENSIONS AND PRODUCTION VOLUMES OF PCP-TREATED WOOD PRODUCTS USED IN CONCENTRATION ESTIMATES

	PER PIECE					
Category	Dimensions (m)	Surface Area (m^2)	Volume (m^3)	Volatilization Volume (m^3)	No Pieces	Total Volatilization Volume (m^3)
Poles	.4 dia x 10 long	12.5	1.25	1.25×10^{-3}	8.3×10^{5}	1.03×10^{4}
Lumber	.1 x .1 x 2.44	1.0	0.25	1×10^{-3}	1.57×10^{7}	1.57×10^{4}
Fenceposts	.2 dia x 2 long	1.25.	0.063	1.25×10^{-3}	4.1×10^{6}	5.17×10^{3}
Crossarms	.1 x .1 x 2.44	1.0	0.025	1×10^{-3}	5.14×10^{6}	5.14×10^{3}
					Total:	3.6×10^{4}

Source: Arthur D. Little, Inc., estimates.

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TABLE 29. ESTIMATED PCP VOLATILIZATION LOSS BY WOOD PRODUCT CATEGORY

Category	Volume From Which PCP Volatilizes (m^3)	PCP Concentration in Wood (kg/m^3)	Total PCP Lost (kg)
Poles	1.03×10^4	12.7	1.3×10^5
Lumber	1.57×10^4	8.1	1.3×10^5
Fenceposts	5.17×10^3	8.1	4.2×10^4
Crossarms	5.14×10^3	8.1	4.2×10^4
		Total	3.44×10^5

Based on assumptions stated previously 2.6×10^4 kg will be dispersed in 4.7×10^{15} m^3 of air, giving a concentration estimate of 5.6×10^{-9} g/m^3. An order of magnitude error either way in this estimate yields a range of approximately 0.6 to 56 ng/m^3.

d. Atmospheric Concentrations due to Evaporation from Cooling Towers

Each year, 2.3×10^5 kg of pentachlorophenol is used in the treatment of cooling tower waters (MITRE, 1979). It is difficult to predict the relative amounts of this material that might be volatilized or be discharged with the cooling water into holding ponds or directly into water bodies after once through the cooling cycle. The conservative assumption is that all of this PCP escapes into the atmosphere, along with the evaporated water and steam from cooling towers.

The previously adopted assumptions regarding atmospheric dilution and emission are assumed. In addition removal processes such as deposition and rainout from the plume are assumed not to occur. Consequently 17250 kg of PCP will be dispersed in 4.7×10^{15} m^3 of air, giving an average concentration estimate of about 3.6 ng/m^3 due to evaporation of PCP in cooling tower waters. Order of magnitude uncertainty in this estimate gives a range from 0.4 ng/m^3 to 36 ng/m^3.

e. Atmospheric Concentrations Due to Open Burning of PCP-Treated Wood Products

No information was found regarding the fraction of the original amount of PCP in wood which is released during open burning. Experiments have been done, however, on the incineration of PCP in a wood-burning furnace of the type used to dispose of wood scrap and sawdust treated with sodium pentachlorophenate from woodworking operations (Ahling and Johansson, 1977). Though these cases may seem to be similar, it is not possible to draw an analogy between the cases of burning in a furnace and open burning.

In a pilot-scale furnace, waste PCP solution was injected into the combustor at 50 to 70 g/hr with high excess air. The PCP residence time was 3.5 seconds and the temperature was ~600°C. 52 mg PCP remained per kg PCP charged to the furnace. At a temperature of 700°C the remainder was 23 mg/kg and at 800°C it was 16 mg/kg. The heat load on the furnace (heat removed that would otherwise contribute to fuel and PCP decomposition) affected the product yield. In a full scale furnace running at 2 to 11 g PCP/hr, PCP remainder was between 1.6 mg/hr (645 mg/kg) at a low load to 79 mg/hr (7100 mg/kg) at a high load. Therefore 0.6% to 0.7% of the original PCP remained in the flue gas. The high PCP emission rates were attributed to excessively high loading, even though temperature drop was moderate. The periods or regions where temperature in the furnace is less than 700°C would account for most of the emission. At 750-800°C, the destruction of PCP is more reliable.

Primary variables in the comparison between furnace incineration and open burning are: initial amount of PCP available for burning (either kgPCP/kg wood or as kgPCP/hr); residence time of PCP in the hot combustion zone; temperature; equivalence ratio; and modes of O_2 transport to the wood surface. Two types of processes are possible. PCP may oxidize, leading to the formation of various combustion products, or it may pyrolyze, a thermal degradation that occurs in the absence of oxygen. It may evaporate unchanged.

With respect to the variables mentioned above, open burning is significantly different from combustion in a furnace. There is likely to be more PCP in wood than was injected into the furnace as a PCP solution. Wood is treated with PCP at concentrations up to 8 kg PCP/m^3 wood or .014 kg PCP/kg wood (pine wood at 554 kg/m^3). In the furnace tests up to 11 g PCP/hr were injected. In an open fire, more than a kg of wood would be burned per hour; thus, more than 11 g PCP/hr would be in the burning wood.

In the open burning case, PCP could remain in regions of high temperature longer than 3.5 seconds since the material is tightly absorbed into the wood and remains at high temperatures for a longer time, both inside the wood, on the glowing surface, and in the flame. It is also quite possible though that PCP may evaporate or boil off quickly and escape.

Temperatures in open burning in some regions may be considerably higher than the 700°C-800°C existing in the furnace. Surface temperatures of burning wood approach 1000°C.

In the furnace tests, much excess air, and therefore oxygen, was available to the burning wood and PCP. This is a significant factor leading to increases of the oxidation in the furnace, thereby reducing the amount of unburned hydrocarbons. Lower availability of oxygen is likely to occur in open burning with unreacted PCP introduced into the air, because of incomplete reaction.

The lack of available oxygen, taken along with the increased amount of PCP in the wood, will tend to decrease the fraction of PCP reacted in open burning. These uncertainties suggest that the furnace test results should not be used to estimate the PCP burned or released in open waste fires.

In order to estimate the atmospheric loading from the release of PCP by open burning, scrap timber, lumber, and plywood are assumed to be the only products burned. Fenceposts, utility poles, and crossarms will not be burned to any great extent, whereas wood scraps from timber, lumber, and plywood of little economic use are likely to be disposed of by burning. Of annual PCP use, 15.6% or 2.6×10^6 kg, goes into the timber, lumber, and plywood category (U.S. EPA 1980b). If 1% of this total is assumed to be disposed of by burning, 2.6×10^4 kg/yr could be put into the air. Conclusions from the discussion above indicate that no quantitative estimates can be made of the PCP actually oxidized or pyrolyzed vs. the amount that is merely evaporated unchanged. The potential, therefore, exists for up to 100% of this 2.6×10^4 kg of PCP to enter the atmosphere each year.

Following previously stated assumptions, the atmospheric concentrations due to open burning are estimated to be ~0.4 ng/m^3. If the assumed amount of scrap burned is increased to 10% of the total 2.6×10^6 kg of PCP use, the atmospheric concentration is increased to 4 ng/m^3. When order of magnitude uncertainty is included, the resulting estimate ranges from 0.04 ng/m^3 to 40 ng/m^3.

f. Ground-Level Concentrations in the Plume Downwind of a Cooling Tower

Pentachlorophenol is used as a biocide in cooling tower waters. Biocides may be injected continuously or used intermittently as a shock treatment (Shair and Dorrington, 1976). It is possible that the PCP may be vaporized from the cooling water and then be dispersed in the atmosphere or that it may be concentrated in the cooling tower blowdown. However, owing to the increased vapor pressure of PCP at higher temperatures (1.6×10^{-4} torr at 25°C and 3.1×10^{-3} at 50°C) and to the higher volatility of PCP in steam and hot water (USEPA, 1978a), it seems reasonable to assume that some, if not all, of the PCP will be emitted into the atmosphere. The conservative assumption adopted is that the relative concentration of PCP in this plume is the same as that in the makeup water.

PCP is an effective biocide at a concentration of about 0.01% (USEPA, 1978c). In this analysis, the water is assumed to be treated with PCP to 100 mg/l. NaPCP is used at concentrations of 5-85 mg/l (USEPA, 1978a) and chlorophenols in general at about 30 mg/l (Glover, 1975). It is possible that higher concentrations may be used in the makeup water; in this case concentrations in the plume will increase by a similar amount.

The most common type of industrial cooling tower is the forced draft, open recirculating type (Matson, 1977). Typical specifications for a cooling tower of this type are shown in Table 30. Water is used in these units as a low-level heat sink and is cooled by evaporation and heat exchange with air in a cooling tower. Makeup water is added to compensate for evaporation and drift, the entrained water droplets in the air leaving the cooling tower. The evaporating water and drift cause the plume.

TABLE 30. TYPICAL OPERATING VALUES FOR AN INDUSTRIAL FORCED-DRAFT, OPEN RECIRCULATING-TYPE COOLING TOWER

Operating Characteristic	Value
Makeup water	34 m^3/min
Blowdown rate	5.7 m^3/min
Evaporation rate	26 m^3/min
Drift rate	0.85 m^3/min
Recirculating water rate	1400 m^3/min
Cooling tower ΔT across tower	11°C
Cooling tower heat load	146×10^6 W

SOURCE: Matson, 1977.

The ground-level concentrations of PCP in the cooling tower plume can be calculated. Since a large amount of heat is released in the plume, it is assumed to be bouyant. The initial height of the plume is assumed to be 10 m. The adjusted source height, due to bouyancy, is given by (Briggs, 1969).

$$h = h_s + 1.6 \left[\frac{gH}{\pi p_a c_p T} \right]^{1/3} U_w^{-1} \; x^{2/3}$$

where

h_s = initial source height, 10m,
g = acceleration of gravity, 9.8 m/sec.
H = heat load of cooling tower, 146×10^6W,
p_a = density of air, 1.23 kg/m^3,
c_p = specific heat, about 1.465 J/kg°K for air/steam,
T = ambient temperature, 300°K,
U_w = wind speed, 3 m/sec, and
x = distance downwind, = $10h_s$ when $x > 10h_s$,

The adjusted source height for the plume becomes, in this case, 120 m. For a Gaussian concentration distribution in the plume, ground level PCP concentration C_c (x,o,o) can be computed from (Slade, 1968):

$$C_c\,(x,\,o,\,o) = \frac{\dot{Q}}{\pi\sigma_y\sigma_z U_w}\left(\exp\left[-\frac{h^2}{2\sigma_z^2}\right]\right)$$

where

$\dot{Q}$ = mass input, 0.045 kg PCP/sec,
σ_y = horizontal dispersion parameter, m.
σ_z = vertical dispersion parameter, m.
U_w = mean wind speed, 3 m/sec, and
h = source height, 10 m or 120 m.

This equation models a dispersing plume being carried along steadily and uniformly by the wind. Dispersion coefficient σ_y and σ_z are dependent on meteorological conditions and downwind distance. Values used are given in Table 31.

TABLE 31. DISPERSION COEFFICIENTS AS FUNCTION OF DOWNWIND DISTANCE (PARTLY SUNNY DAY, WIND 3 M/SEC)

x, km	0.3	0.5	0.7	1.0	3.0	5.0	7.0	10.0
σ_y, m	50	84	112	150	375	670	890	1200
σ_z, m	30	51	73	110	360	650	920	1400

Source: Slade, 1968.

Values of PCP concentration at ground level downwind of a cooling tower were computed for source heights of 10 m and 120 m (buoyant plume) and the plots of the results are shown in Figure 13. Maximum concentrations appear within a kilometer or two of the tower and then decrease with distance. Many other factors will affect the dispersion process, including rainout, terrain induced dispersion, large-scale atmospheric turbulence, and possible reactivity in the plume. These factors, which have not been modelled, would tend to reduce concentrations.

The results of the computations can be scaled linearly in order to reflect increased or decreased PCP concentrations in the makeup water. Values from Figure 13 should then be adjusted accordingly.

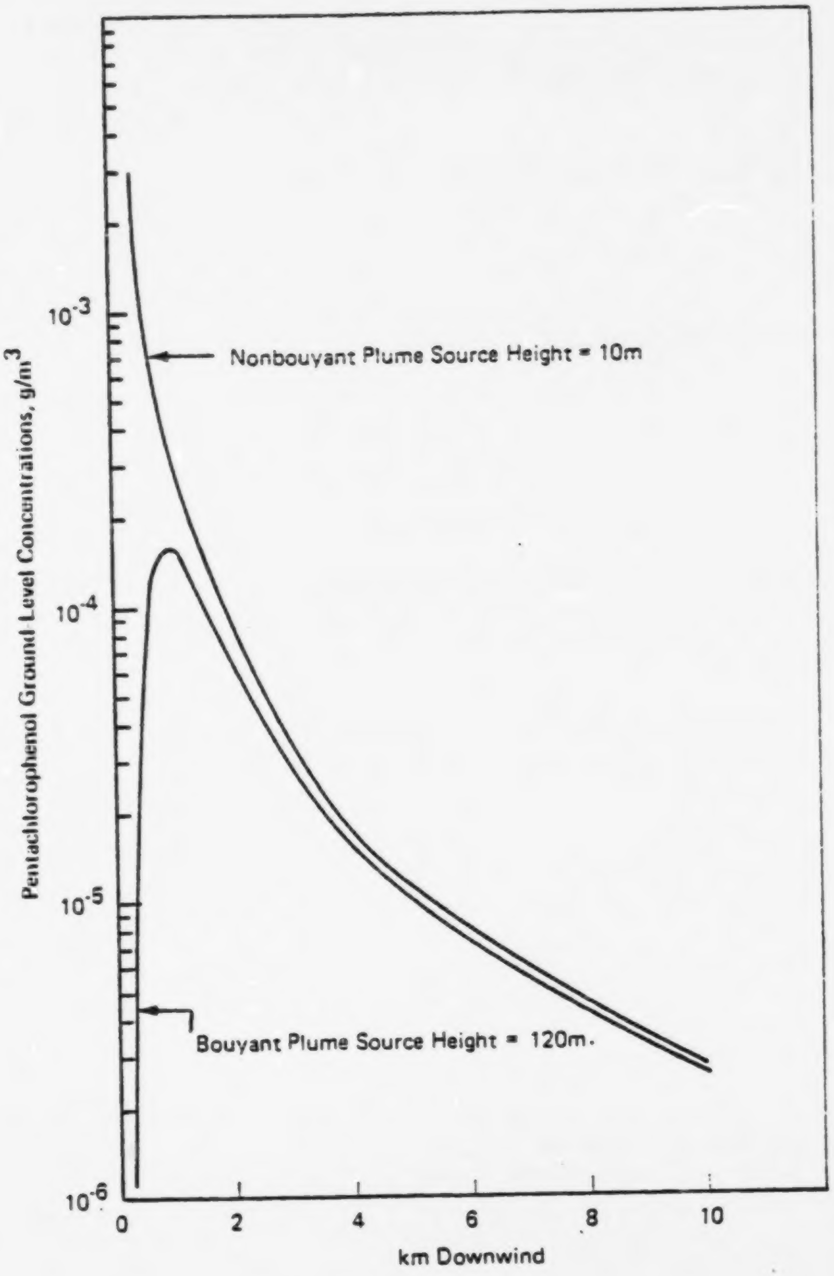

FIGURE 13 GROUND-LEVEL CONCENTRATIONS OF PENTACHLOROPHENOL IN THE PLUME DOWNWIND OF A COOLING TOWER – TWO SOURCE HEIGHTS

The concentration estimates in a plume downwind of a cooling tower on the order of 10^4 ng/m^3 are considerably higher than those estimated for other particular atmospheric inputs as compared to 10 ng/m^3

g. Concentrations Downwind of a Wood Treatment Plant Waste Water Evaporation Pond

Wood treatment plants treat effluent and then discharge this treated waste into evaporation ponds to achieve zero effluent discharge into streams and other water bodies (USEPA, 1979a and 1979b). This effluent contains pentachlorophenol. As the water in the ponds evaporates, some of this PCP may evaporate along with the water. Although volatilization appears to be a minor fate process, another pathway for removal of PCP from pond water is adsorption to sludge or sediment in the bottom of the pond. Some ponds are equipped with mechanical aerators to enhance PCP and water evaporation. For this analysis, the conservative assumption is adopted that all the PCP in the waste water evaporates into the air in order to obtain an upper limit for air releases and to compute PCP concentrations in the plume downwind from an evaporation pond.

The ponds are designed to contain all process water used by a plant in addition to water that may enter the plant site through precipitation; therefore, in continuous operation, these ponds must be capable of evaporating water at least at the rate it is used by the plant. Therefore, the daily water use is assumed to be evaporated as it is used. As an example, a daily process water throughput of 3.8×10^5 l is assumed. Furthermore, this water will contain 2.8 mg/l PCP (Branson and Blau, 1979) when it is discharged into a 4×10^4 m^2 pond. Thus, in steady-state operation, 1.23×10^{-5} kg PCP/sec is evaporating.

Concentrations downwind from an area source discharging continuously into the air can be estimated (Slade, 1968) assuming a virtual point source located five area source diameters upwind. For the example chosen, the diameter of the source is $\sim$220m and the virtual point source is 1.1 km upwind. Standard Gaussian dispersion formulas can be employed to estimate centerline ground-level concentrations downwind from a surface source. The equation is (Slade, 1968):

$$C(x,o,o) = \frac{\dot{Q}}{\pi\sigma_x\sigma_y U_w}$$

where

$\dot{Q}$ = release rate of pollutant, 1.23×10^{-5} kg/sec,
σ_y = horizontal dispersion coefficient
σ_z = vertical dispersion coefficient, and
U_w = wind speed, 3 m/sec.

based on virtual source 1.1 km upwind and the assumed stability conditions

Dispersion coefficients used are shown in Table 32. The results of the calculations are shown in Figure 14.

TABLE 32. DISPERSION COEFFICIENTS USED IN THE CALCULATIONS OF CONCENTRATIONS DOWNWIND OF AN EVAPORATION POND RELEASING PCP INTO THE AIR

	Dispersion Coefficient[1]				
Actual km downwind	.5	1	2	3	4
Model km downwind	1.6	2.1	3.1	4.1	5.1
σ_y, m	240	310	430	570	700
σ_z, m	175	240	370	510	670

[1]Based on partly sunny day; 3 m/sec wind.

Source: Slade, 1968.

Terrain induced dispersion, large scale atmospheric turbulence, and rainout or deposition are neglected in the calculations, thereby giving upper limit concentrations in the ng/m^3 range at distances greater than 1 kilometer.

h. Rainout

Dissolution in water droplets and subsequent rainout is considered here as a possible atmospheric loss mechanism. PCP has been found in rainwater in Hawaii at concentrations of about 8 ng/1-284 ng/1 (Bevenue *et al*., 1972b) (see Table 6). Concentrations of PCP in rainwater can be estimated from a knowledge of concentration in air and the non-dimensional Henry's Law coefficient . For equilibrium conditions (Slinn *et al*., 1978):

$$C_{rain} = \frac{C_{air}}{H}$$

where

C_{rain} = concentration in rainwater,

C_{air} = concentration in air, and

H = non-dimensional Henry's Law coefficient.

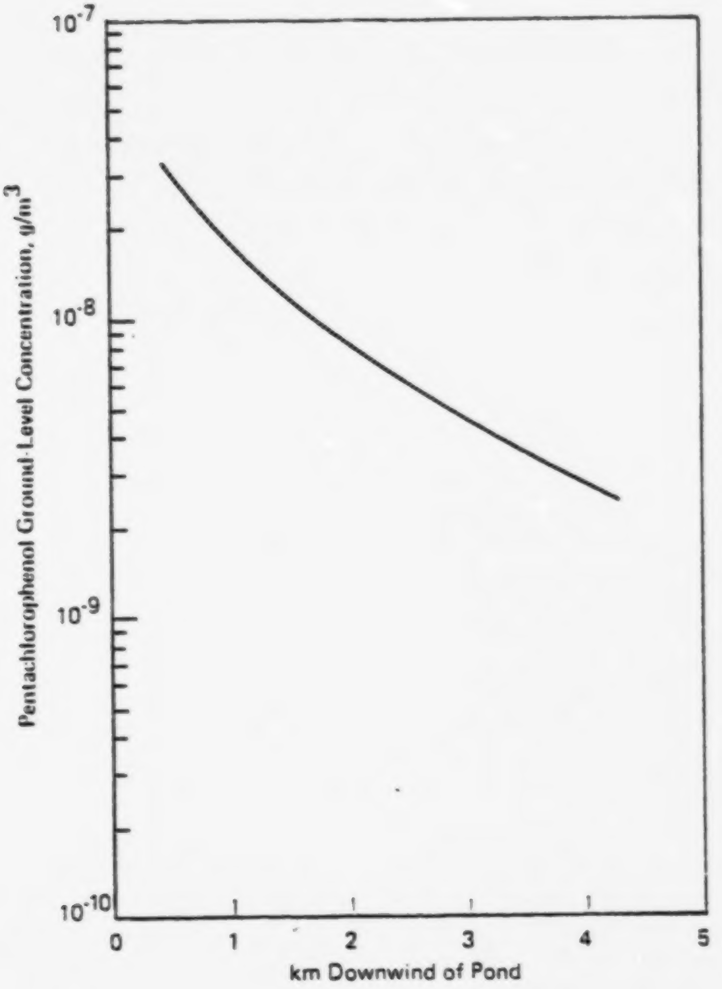

FIGURE 14 GROUND-LEVEL PENTACHLOROPHENOL CONCENTRATIONS IN THE PLUME DOWNWIND OF A WOOD TREATMENT PLANT EVAPORATION POND

H can be computed from (Neely, 1976):

$$H = \frac{P_v}{P_t} \frac{M}{29} \frac{\rho_{air}}{X_s}$$

where

P_v = vapor pressure of PCP at 20°C, 1.7×10^{-4} torr, upper value of range reported,*

P_t = partial pressure or air, 760 torr (1 atm.),

M = molecular weight of PCP, 266.35,

29 = average molecular weight of air,

ρ_{air} = density of air, 1.23 kg/m^3, and

X_s = solubility of PCP, .014 g/l = 0.014 kg/m^3*.

The non-dimensional H is then computed to be 1.8×10^{-4}, and

$$C_{rain} = 5556 \, C_{air}$$

For equilibrium between water and air, the air concentration required to cause a concentration of 8-284 ng/l in rainwater is, using the values of the Henry's Law coefficient computed above, between 0.002 to 0.063 ng PCP/m^3 air. The cumulative air concentration estimated from release rates of PCP from burning, volatilization from treated produced wastewater evaporation, and cooling tower use is between 0.5 and 136 ng/m^3. The ranges for the inputs were 0.04 to 40, 0.06 to 56, 0.005 to 4.3, and 0.4 to 36 ng/m^3, respectively. The air concentrations estimated from rain data are somewhat lower than the cumulative estimate, though the rain-based estimate still falls partially in the lower range of the cumulative estimate.

PCP concentration in the rain in Hawaii was attributed to the large use of PCP-treated building materials. The general agreement noted here tends to support the validity of the estimates derived above, especially considering that the estimated concentration based on volatilization from treated products was 0.06 to 56 ng/m^3, which is quite similar to the estimate based on rain data.

i. Conclusions

Inputs to the air compartment seem to occur steadily over a large area. Estimates were derived of PCP input to air from evaporation of wood treatment facility waste water, volatilization from treated wood products, PCP-treated cooling tower water evaporation, and open burning of PCP-treated wood. Total inputs to the air were estimated as 3.0 x 10^3 to 2.4 x 10^4 kg/yr, 3.4 x 10^5 kg/yr, 17250 kg/yr, and 2.6 x 10^4 to 2.6 x 10^5 kg/yr, respectively. This produced concentration estimates due to each source of 0.005 - 4.3, 0.06 - 56, 0.04 - 36, and 0.04 0.40 ng/m^3, respectively. A cumulative concentration estimate is then 0.15 to 136 ng/m^3. The lower range of this estimate is in general agreement with the upper range of air concentration estimates computed based on PCP concentrations in rain in Hawaii, where PCP treated wood is extensively used. This estimate gave 0.002 to 0.063 ng/m^3.

In addition, ground level concentrations in the plume downwind from a cooling tower using PCP as a microbiocide and downwind from a waste water evaporation pond were computed. Estimates were about 10^5 ng/m^3 and 10 ng/m^3, respectively, for distances less than a few kilometers.

3. Water Models

a. Introduction

The EXAMS model has been developed by EPA Athens Environmental Research Laboratory in order to help assess the behavior of a pollutant in various characteristic aquatic systems (U.S. EPA 1980c). The output of the model includes:

(1) simulations of steady-state concentrations and pollutant mass distribution among water, sediment and biota in different compartments (e.g. water column, bed sediment),

(2) percentage of system-loading removed by each chemical and biological kinetic process, and

(3) concentration die-away time following cessation of discharge.

As input the model requires the pollutant's physico chemical properties, environmental reaction rate constants, and loading rate to the system. The assumptions of the model include a continuously discharging source at a constant level, a box of water made up of a system-defined number of well-mixed compartments, and first-order rate kinetics in all processes. A more thorough discussion of the model is given in Lassiter, et al. (1978) and Baughman and Burns (1980).

Input data for PCP was derived from two sources. The chemical property and rate data[1] were compiled specifically for the model by EPA (Versar, 1980). The loading data were derived from effluent concentration and flow data characteristic of various industry categories that discharge PCP. Table 33 lists upper limit PCP loads calculated from effluent guidelines discharge data. The data can not be assumed to represent worst case discharge levels for each industry because they are derived from a small number of plants and days sampled. On the other hand, unless other information was available, the assumption was made that the sampled concentration and flow data represent practices typical

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[1] Properties were for the undissociated molecule. PCP

TABLE 33. ESTIMATES OF UPPER LIMIT PCP DAILY LOAD IN INDUSTRIAL EFFLUENTS[1]

Industry	Flow (MGD)[2]	PCP Concentration (mg/l)	Daily Load (kg/day)
Leather Tanning	0.300	15.0	17.1
Textiles	1.0	0.066	0.25
Wood Preserver	0.014[3]	20	0.07[4]

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[1] Loadings are estimates of upper limit amounts based on a very limited sample of data from effluent guidelines.

[2] MGD = million gallon per day

[3] Average number of days of discharge per year = 10 - 25 days.

[4] Discharge averaged out over entire year.

for every day of the year. This may result in too high a loading rate but, until more detailed discharge data become available, this assumption is justified.

A major drawback of using the EXAMS model for PCP relates back to the discharge practices of the pollutant's major users. Only one wood preserving plant practices direct discharge and the other industry category dischargers account for a relatively small amount of the PCP pollutant load. Most sources are non-point sources and estimating their release rates is difficult. Therefore, the results of the model apply only to a small number of known situations and cannot be assumed to be representative of widespread conditions.

b. EXAMS Concentration Estimates

Tables 34-37 present the output from the EXAMS model (U.S. EPA 1980c). Steady-state maximum concentrations of PCP in the six different components of each aquatic system are listed for the three discharge rates of 17.1 kg/day (Table 34), 0.25 kg/day (Table 35) and 0.07 kg/day (Table 36). Table 37 presents the percentage of total PCP loss attributable to each transformation or transport process and the estimated time for self-purification for each of the six aquatic systems. These values are independent of loading rate but dependent on the physico-chemical characteristics of PCP and system characteristics.

In general, the results show higher concentration accumulation in the water compartments of the pond and two lake systems than in the three river systems. Suspended sediment concentrations are approximately 2 to 2.5 orders of magnitude greater than dissolved concentrations in water. Bottom sediment concentrations are similar to water concentrations, sometimes slightly greater and sometimes less than dissolved water levels, depending on the particular system. Plankton concentrations are approximately 15 to 20 times above dissolved water concentrations.

Table 37 describes the steady-state distribution of PCP between water and sediment, and the percentage transformed by each process. Physical transport processes account for almost all losses in the river systems and for most of the loss from the pond and oligotrophic lake systems. According to the model, biodegradation is very significant in the eutrophic lake system. Chemical processes account for one-third of the PCP disappearance in the oligotrophic lake system. The estimated time for self-purification ranged from 96.64 days in the river system to 695.5 days in the oligotrophic lake. Persistence is a complex phenomenon dependent on many site-specific non-generalizable variables. All of these values are merely estimates and may not be indicative of the lifetime of PCP in specific natural aquatic ecosystems.

Further discussion of the EXAMS concentration simulations is found in Sections VI-B and VII-C.

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TABLE 34

STEADY-STATE CONCENTRATIONS IN VARIOUS GENERALIZED AQUATIC SYSTEMS RESULTING FROM CONTINUOUS PCP DISCHARGE AT 17.1 kg/day[1]

SYSTEM	LOADING	MAXIMUM CONCENTRATIONS						TOTAL STEADY-STATE ACCUMULATION (kg)	TOTAL DAILY LOAD (kg/day)
		WATER DISSOLVED (mg/l)	WATER TOTAL (mg/l)	BOTTOM SEDIMENT (mg/l)	BOTTOM SEDIMENT (mg/kg)	PLANKTON (µg/g)	BENTHOS (µg/g)		
Pond	(0.713 kg/hr) (17.1 kg/day)	21.38	26.36	0.249	78.38	3.92×10^{5}	4.49×10^{3}	583.33	17.1
Eutrophic Lake		0.121	0.228	5.56×10^{-3}	0.235	2.14×10^{3}	99.75	420.42	17.1
Oligotrophic Lake		0.584	1.14	1.43×10^{-2}	3.06	1.07×10^{4}	249.38	3240.0	17.1
River		7.04×10^{-4}	7.84×10^{-4}	1.71×10^{-4}	4.63×10^{-2}	12.83	3.06	0.784	0.713
Turbid River		7.04×10^{-4}	7.84×10^{-4}	3.69×10^{-4}	4.42×10^{-3}	12.83	6.48	0.784	0.713
Coastal Plain River		6.91×10^{-3}	7.84×10^{-3}	5.99×10^{-3}	9.25×10^{-2}	128.25	37.75	7.840	0.713

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[1] All data simulated by EXAMS model (see text for further information).

TABLE 35

STEADY-STATE CONCENTRATIONS IN VARIOUS GENERALIZED AQUATIC SYSTEMS RESULTING FROM CONTINUOUS PCP DISCHARGE AT 0.25 kg/day[1]

		MAXIMUM CONCENTRATIONS							
SYSTEM	LOADING	WATER DISSOLVED (mg/1)	WATER TOTAL (mg/1)	BOTTOM SEDIMENT (mg/1)	MAXIMUM IN DEPOSITS SEDIMENT (mg/kg)	PLANKTON (µg/g)	BENTHOS (µg/g)	TOTAL STEADY-STATE ACCUMULATION (kg)	TOTAL DAILY LOAD (kg/day)
Pond	1.04×10^{2} kg/hr (0.25 kg/day)	0.313	0.38	3.65×10^{-3}	1.15	5.73×10^{3}	65.63	8.54	0.25
Eutrophic Lake		1.8×10^{-3}	3.33×10^{-3}	8.13×10^{-5}	3.42×10^{-3}	31.25	1.46	6.17	0.25
Oligotrophic Lake		8.5×10^{-3}	1.67×10^{-2}	2.08×10^{-4}	4.48×10^{-2}	156.25	3.65	50.0	0.25
River		1.03×10^{-5}	1.14×10^{5}	2.50×10^{-6}	6.79×10^{-5}	0.188	4.5×10^{-2}	1.17×10^{-2}	1.04×10^{-2}
Turbid River		1.03×10^{-5}	1.15×10^{-5}	5.21×10^{-6}	6.46×10^{5}	0.188	9.5×10^{-2}	1.17×10^{-2}	1.04×10^{-2}
Coastal Plain River		1.01×10^{-4}	1.15×10^{-4}	3.02×10^{-5}	1.35×10^{-3}	1.88	0.552	0.115	1.04×10^{-2}

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[1] All data simulated by EXAMS model (see text for further information).

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TABLE 36

STEADY-STATE CONCENTRATIONS IN VARIOUS GENERALIZED AQUATIC SYSTEMS RESULTING FROM CONTINUOUS PCP DISCHARGE AT 0.07 kg/day[1]

SYSTEM	LOADING	MAXIMUM CONCENTRATIONS						TOTAL STEADY-STATE ACCUMULATION (kg)	TOTAL DAILY LOAD (kg/day)
		WATER DISSOLVED (mg/1)	WATER TOTAL (mg/1)	BOTTOM SEDIMENT (mg/1)	MAXIMUM IN DEPOSITS SEDIMENT (mg/kg)	PLANKTON (µg/g)	BENTHOS (µg/g)		
Pond	2.92×10^{-3} kg/hr (0.07 kg/day)	8.88×10^{-2}	0.108	1.02×10^{-3}	0.321	1.60×10^{3}	18.38	2.39	0.07
Eutrophic Lake		7.92×10^{-5}	9.33×10^{-4}	2.28×10^{-5}	9.58×10^{-4}	8.75	0.408	1.72	0.07
Oligotrophic Lake		2.39×10^{-3}	4.67×10^{-3}	5.83×10^{-5}	1.25×10^{-2}	43.75	1.02	14.0	0.07
River		2.89×10^{-6}	3.21×10^{-6}	7.00×10^{-7}	1.90×10^{-5}	5.25×10^{-2}	1.25×10^{-2}	3.33×10^{-3}	2.92×10^{-3}
Turbid River		2.89×10^{-6}	3.21×10^{-6}	1.46×10^{-6}	1.79×10^{-5}	5.25×10^{-2}	2.67×10^{-2}	3.33×10^{-3}	2.92×10^{-3}
Coastal Plain River		2.83×10^{-5}	3.21×10^{-5}	8.46×10^{-6}	3.79×10^{-4}	0.525	0.155	3.21×10^{-2}	2.92×10^{-3}

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[1] All data simulated by EXAMS model (see text for further information).

TABLE 37. THE FATE OF PCP IN VARIOUS GENERALIZED AQUATIC SYSTEMS[1]

System	% of PCP Residing in Water at Steady-State	% of PCP Residing in Sediment at Steady-State	% Transformed by Chemical Processes	% Transformed by Biological Processes	% Volatilized	% Lost by Other Processes[2]	Time for System Self-Purification (day)[3]
Pond	90.62	9.38	6.05	12.84	0	81.11	158.3
Eutrophic Lake	98.87	1.13	3.99	88.69	0	7.32	103.9
Oligotrophic Lake	98.99	1.01	33.31	0.09	0	66.41	695.5
River	93.98	6.02	0.01	0.09	0.19	99.9	111.7
Turbid River	94.84	5.16	0.01	0.09	0	99.9	96.64
Coastal Plain River	85.19	14.81	0.09	0.92	0	98.99	580.7

[1] All data simulated by the EXAMS model (see text for further information).

[2] Including loss through physical transport out of system.

[3] Estimate for removal of ca. 75% of the toxicant accumulated in system. Estimated from the results of the half-lives for the toxicant in bottom sediment and water columns, with overall cleansing time weighted according to the toxicant's initial distribution.

4. Summary of Concentration Estimates

Through use of simple calculational models for air and a computerized fate model for water, estimates were made of concentrations in these media resulting from PCP emissions. The results are of two general types: 1) concentrations by source category evenly distributed across the United States or large U.S. regions that represent the sum of contributions from numerous point sources; and 2) concentrations for local sites in the vicinity of single point sources. Tables 38 and 39 summarize the concentration estimates, regional and local, respectively.

TABLE 38. SUMMARY OF ESTIMATES OF REGIONALLY DISTRIBUTED PCP CONCENTRATIONS IN ENVIRONMENTAL MEDIA

Source	Medium	Concentration
Wood Treatment Waste Water Evaporation	Air	4.3 ng/m^3 [1]
Volatilization From Treated Wood	Air	56.0 ng/m^3
Evaporation From Cooling Towers	Air	36.0 ng/m^3
Open Burning Of Treated Wood	Air	40.0 ng/m^3
Summation Of All Sources	Air	136.3 ng/m^3

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[1] All estimates are upper limit concentrations.

TABLE 39. SUMMARY OF NEAR-FIELD ESTIMATES OF PCP CONCENTRATIONS IN ENVIRONMENTAL MEDIA

Source	Medium	Concentration
Cooling Tower Plume	Air	$\sim 10^4$ ng/m^3(1)
Waste water Evaporation Pond	Air	~ 10 ng/m^3
Any source:		
0.07-17.1 kg/day discharge	Pond	107.9 - 26362 μg/ℓ [2]
0.07-17.1 kg/day discharge	Eutrophic Lake	0.1 - 227.9 μg/ℓ
0.07-17.1 kg/day discharge	Oligotrophic Lake	0.005 - 1.4 μg/ℓ
0.07-17.1 kg/day discharge	River	0.003 -0.784 μg/ℓ
0.07-17.1 kg/day discharge	Turbid River	0.003 - 0.784 μg/ℓ
0.07-17.1 kg/day discharge	Coastal Plain River	0.032 - 7.84 μg/ℓ 0.032 - 7.84 μg/ℓ

[1] Upper limit concentrations.

[2] Range of maximum values in total water compartment of EXAMS.

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SECTION V.

HUMAN EFFECTS AND EXPOSURE

A. EFFECTS ON HUMANS

1. Introduction

Pentachlorophenol (PCP) is an antimicrobial agent widely used for the preservation of wood. Most commercial PCP formulations contain from 85% to 99% PCP, as well as different chlorophenol impurities and minor amounts of highly toxic polychlorinated dibenzo-p-dioxins and dibenzofurans. The quantity and proportion of contaminants in the technical product differ somewhat from batch to batch but generally include: tetrachlorophenol (5-10%), trichlorophenol (1%), chlorinated phenoxyphenols which act as dioxin precursors (∿5%) and polychlorinated dibenzo-p-dioxins and dibenzofurans (range 10-1000s mg/kg) (Report of the Ad Hoc Study Group on Pentachlorophenol Contaminants, 1978; Nilsson et al., 1978). The most toxic dioxin known, 2,3,7,8-tetrachlorodibenzo-p-dioxin, has not been found in commercial PCP formulations. The presence of toxic contaminants in commercial PCP formulations obfuscates the true toxic effects of PCP itself. However, the few studies available that have toxicologically characterized purified PCP suggest that PCP is a toxic chemical in its own right.

2. Metabolism and Bioaccumulation

The pharmacokinetics of pentachlorophenol have been studied in man, monkey, rabbits, rats and mice. In man, the half-lives for absorption and elimination from plasma or an oral dose of 0.1 mg PCP/kg were 1.3 ± 0.4 hr and 30.2 ± 4.0 hr, respectively. Approximately 74% and 12% of the dose were eliminated in urine within 7 days as PCP and PCP-glucuronide. An additional 4% of the dose was excreted in feces as PCP and its glucuronide (Braun et al., 1978).

A similar pattern is observed in rats. Larsen and co-workers (1972) reported excretion of 68% of an orally administered dose of ^{14}C-PCP in urine and 9-13% in feces of rats within 10 days of dosing. Tissue analysis at 40 hours post-exposure showed highest ^{14}C-activity present in liver (0.23% of administered dose), kidney (0.18%) and serum (∿ 0.15%).

In another study, oral administration of 10 or 100 mg/kg ^{14}C-PCP to rats resulted in the excretion of 80 and 64% of the respective doses in urine. At the 100-mg/kg dose, ^{14}C-activity in urine consisted of unchanged PCP (75%), tetrachlorohydroquinone (15.6%) and PCP-glucuronide (9.4%). The overall elimination of radioactivity was biphasic in both sexes at the lower dose and in males given 100 mg/kg PCP; elimination was monophasic in females at the higher dose (Braun and Sauerhoff, 1976). A similar response was observed following intraperitoneal administration of PCP to rats (Ahlborg, 1978).

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Jakobson and Yllner (1971) reported excretion of both unchanged and conjugated PCP in the urine (72-83% of the dose) and feces (8-21%) of female NMRI mice within 4 days of intraperitoneal or subcutaneous administration of ^{14}C-PCP. The only breakdown product detected was unconjugated tetrachloro-p-hydroquinone.

In rabbits dosed orally with ^{14}C-PCP-Na, considerable amounts of PCP-glucuronide plus small amounts of chloranil and free PCP were detected in urine (Tashiro et al., 1970).

Unlike man, rodents, and rabbits, the rhesus monkey excretes essentially all (68-78%) of an orally administered ^{14}C-PCP dose in urine as unchanged PCP. An additional 12-20% of the dose is excreted in feces and most probably reflects biliary excretion and enterohepatic circulation of PCP. The half-life values for elimination of PCP from plasma were 83.5 hours for females and 72 hours for males, while half-life values for urinary excretion were 92.4 and 40.8 hours, respectively, indicating sex-related differences in the plasma clearance and urinary excretion of PCP in rhesus monkeys (Braun and Sauerhoff, 1976).

Thus, the pharmacokinetic profile of PCP in monkeys is different from that in man, rodents and rabbits. Humans, rats and mice excrete PCP more rapidly than monkeys; in addition, rats and mice metabolize PCP to tetrachlorohydroquinone and PCP-glucuronide while monkeys excrete it as unchanged PCP in the urine. The rat model, therefore, appears to more closely reflect the pharmacokinetics of PCP in man.

Contamination of human tissues with pentachlorophenol appears to be ubiquitous in industrialized societies. Sources of PCP residues in human tissues have not been conclusively established. Biotransformation of hexa- and pentachlorobenzene to PCP may contribute to the contamination of human tissues with PCP (Koss and Koransky, 1978).

In an analysis of 416-418 human urine samples from the general population of the United States, 84.8% were positive for pentachlorophenol. The average residue detected was 6.3 μg/l with a maximum detected value of 193 μg/l (Kutz et al., 1978). The limit of detection was 5 μg/l. Other investigators have reported urinary residues of up to 50 μg/l in the general population (Bevenue et al., 1967; Akisada, 1965).

Urinary PCP residues in 271 occupationally exposed individuals ranged from 3 μg/l to 35700 μg/l, with a mean value of 1244 μg/l (Bevenue et al., 1967). The urine of PCP-wood-treaters contained an average of 2300 μg/l PCP over a one-year period. A mean decrease of 60% (range 39-79%) in urinary concentration was noted in six of these workers 18 days after cessation of work-related exposure (Casarett et al., 1969). Similarly, Begley et al. (1977) reported that blood (5.14 mg/l) and urine (1.31 mg/l) content of PCP decreased by more than 50% in 18 occupationally exposed individuals during a 20-day vacation period and subsequently increased again with renewed work exposure.

PCP content of 18 adipose tissue samples from the general population ranged from 12 to 52 µg/kg with a mean value of 26 µg/kg (Shafik, 1973). Ohe (1979) noted slightly higher residue levels of PCP in human adipose tissue of the general population in Japan; i.e., the PCP content of 25 samples ranged from undetectable to 570 µg/kg, with a mean value of 140 µg/kg.

PCP has also been detected in seminal fluid. Levels detected averaged 50 µg/l (range 20-70 µg/l) (Dougherty and Piotrowska, 1976).

3. Animal Studies

a. Carcinogenesis

Innes *et al*. (1969) administered technical pentachlorophenol (Dowicide 7) by gavage to two hybrid strains of mice [(C57BL/6 x C3H/Anf) F_1 and (C57BL/6 x AKR F_1] at doses of 46.4 mg/kg on days 7-28 of age and at 130 mg/kg (17 mg/kg/day) in the diet of animals up to 18 months of age. No significant increase in tumor incidence above control values was observed. Schwetz *et al*. (1978) also noted no alteration in tumor incidence or duration of lifespan in rats fed up to 30 mg/kg/day of purified PCP for 22-24 months.

In a tumor promotion study, 20% PCP in benzene was applied to the backs of dimethylbenzanthracene-initiated mice twice weekly for 15 weeks. Survival rates for PCP- and benzene-treated mice were 82.9% and 75%, respectively. The average number of papillomas per survivor was 0.04 for PCP-treated group compared with 0.07 papillomas in the control group (Boutwell and Bosch, 1959).

b. Mutagenesis

The literature contains conflicting reports concerning the mutagenicity of pentachlorophenol. Andersen *et al*. (1972) reported that PCP was incapable of inducing point mutations in eight histidine-requiring mutants of *Salmonella typhimurium*. These tests did not employ activation using liver microsomes. PCP also produced negative mutagenic responses in a host-mediated assay (Buselmaier *et al*., 1973) and in a sex-linked recessive test with Drosophila (Vogel and Chandler, 1974).

Fahrig and associates (1978), however, reported a significant increase in forward mutations and mitotic gene conversions in the yeast, *Saccaromyces cerevisiae* MP-1, following exposure to 400 mg/l PCP for 3.5 hr. No positive controls were run. These investigators also examined the expression of recessive color genes in a mammalian "spot" test. Progeny of female C57BL/6JHan mice mated to rotation bred T-stock males are susceptible to color spots in the adult coat if exposed *in utero* to a mutagenic agent. The incidences of color spots in progeny were 1.3% (2/157), 0.6% (2/316) and 0.1% (1/967) from dams injected with 100, 50 or 0 mg/kg PCP on the tenth day of gestation. The statistical significance of these data the incidence of treatment-related maternal toxicity and control results were not stated making interpretation of the results difficult.

Thus, although there are conflicting reports on the mutagenic potential of PCP in the literature, the data currently available are insufficient to support the mutagenicity of PCP.

c. Reproductive and Fetotoxic Effects

In a series of experiments, Schwetz et al. (1974) examined the effects of purified (>98%) and commercial grade (88.4%) PCP[1] on rat embryonal and fetal development. In one study, dosages of 5, 15, 30 and 50 mg/kg/day of purified PCP and 5.8, 15, 34.7 and 50 mg/kg/day of commercial grade PCP were administered by gavage to pregnant Sprague Dawley rats on days 6-15 of gestation. At the 5 mg/kg and 30 mg/kg dosage levels, the amounts of commercial PCP administered (5.8 and 34.7 mg/kg) were adjusted in order to provide 5 mg/kg and 30 mg/kg of PCP.

Dose-related decreases in maternal body weight gain were noted with both PCP samples and were statistically significant at the top two treatment levels. No other signs of maternal toxicity were noted. In progeny, purified PCP exerted a more pronounced effect than commercial grade PCP. Fetal resorptions were increased with both grades of PCP and were statistically significant ($p < 0.5$) in dams given 15, 34.7 or 50 mg/kg/day of commercial PCP or 30 or 50 mg/kg/day of purified PCP. Resorption rates. were 9, 27 and 58%, respectively, for commercial PCP and 98 and 100%, respectively, with purified PCP compared to 4.2% resorptions in controls. The no-effect dosages for fetal resorptions were 5.8 mg/kg/day commercial and 15 mg/kg/day of purified PCP.

The sex ratio of surviving offspring was significantly altered among dams receiving 30 mg/kg (purified) and 50 mg/kg (commercial) PCP; the majority of survivors were males. (LD_{50} is higher in males than females.) Decreases in fetal body weight were seen in rats in the 34.7 and 50 mg/kg/day commercial PCP treatment groups and in the 30 mg/kg purified PCP group; crown-rump length was also decreased in the latter group. There were no survivors among the animals that received 50 mg purified PCP/kg/day. Dosages of 15 mg/kg/day or less of either grade had no effect on fetal body measurements.

The incidences of anomalies in fetuses of dams treated with either grade of PCP on days 6-15 of gestation were not significantly different from control. However, the occurrence of subcutaneous edema, dilated ureters, and anomalies of the skull and vertebrae increased with increasing PCP dose.

However, a second experiment by these investigators (Schwetz et al., 1974) showed that the developing rat embryo was more susceptible to the toxic effects of a given dose of PCP during the period of early organogenesis (days 8-11). Pregnant rats were administered 0 to 30 mg/kg/day of purified or 34.7 mg/kg/day of commercial PCP on days 8 through 11 or days 12 through 15 of gestation. Both samples at the given dose of PCP caused a significant decrease in the rate of maternal weight gain and a significant increase in the incidence of fetal resorptions (45%

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[1] Including 4 ug/g hexachlorodibenzo-p-dioxin, known to be toxic to the rat embryo and fetus (Schwetz et al. 1974).

commercial, 91% purified compared to 7% control) when given on days 8 through 11 of gestation but had no effect on these two parameters when administered on days 12 through 15 of gestation. In addition, significant incidences of fetal anomalies were associated with the administration of PCP during early organogenesis: subcutaneous edema (82% commercial, 100% purified compared to 7% control) as well as skeletal anomalies in the development of ribs, vertebrae and sternebrae. No increase in anomalies was associated with the administration of commercial PCP during late organogenesis (days 12-15). Administration of purified PCP during this period, however, resulted in a significant increase in the incidence of subcutaneous edema (70% vs. 36% in controls) and variations in sternebral development.

Schwetz and co-workers (1978) also conducted a single generation reproduction study with Sprague Dawley rats. Animals were fed 0, 3 or 30 mg/kg/day of purified PCP for 62 days prior to mating, throughout gestation and up to 21 days postpartum. Ingestion of 30 mg/kg resulted in a significant decrease in the percentage of pups that were born alive (91 versus 97% for controls) and significantly decreased survival to days 7, 14 and 21 of lactation,but had no effect on fertility. A significantly increased number of litters showed skeletal anomalies at this dose, the average litter size was decreased, and mean neonatal body weight was significantly less than controls. No adverse effects were noted at doses of 3 mg/kg/day.

Comparable effects were not observed when a larger (60 mg/kg), but single oral dose of purified PCP was given to pregnant Charles River CD strain rats on either day 8, 9, 10, 11, 12 or 13 of gestation. Body weight of fetuses from dams treated on days 9 or 10 was significantly reduced and three malformations were observed in 3 of 59 fetuses treated on days 9 of gestation (Larsen _et al_., 1975).

Chou and Cook (1979) reported recently in a meeting abstract that intraperitoneal injection of either 0, 0.4, 4 or 40 mg/kg of technical PCP or purified PCP adjusted to provide the same level of PCP to pregnant Sprague Dawley rats on days 3, 6, 9, 12 and 15 of gestation resulted in increased fetal resorptions and lower maternal weight gain in dams treated with the highest dosage of either sample of PCP. The effect was reportedly more pronounced with purified PCP. No values were given. No teratogenic response was noted and performance of pups was comparable to control values.

Hinkle (1973) reported that daily oral administration of 1.25 mg/kg to 20 mg/kg PCP to golden Syrian hamsters on days 5 to 10 of gestation resulted in fetal deaths and/or resorptions in three of six test groups. No data were provided.

In summation, fetotoxic and teratogenic effects have been produced in rats following oral administration of 30 mg/kg/day or greater of pentochlorophenol during gestation. Purified PCP was somewhat more toxic than commercial grade PCP with respect to the incidence of fetal resorptions, growth retarded fetuses, and the incidence of skeletal and soft tissue anomalies, particularly during early organogenesis. Comparable effects were not observed following a single but higher oral dose of PCP.

d. Other Toxic Effects

Toxicological findings concerning PCP are complicated by the presence of varying amounts of contaminants in the technical material. The oral LD_{50} (50% lethal dose) for PCP in male rats has been reported as 78 mg/kg (Deichmann et al., 1942), 90 mg/kg (Gabrilevskaya and Laskina, 1964), 146 mg/kg (Gaines, 1969) and 205 mg/kg (Schwetz et al., 1974) The last value was for a purified sample of PCP; the LD_{50} in female rats for this material was 135 mg/kg, a finding indicating male rats are more tolerant of the acute toxic effects of PCP than are female rats. Age-related differences were also noted between adult (150 mg/kg) and 3-4-day-old rats (65 mg/kg) with a commercial PCP sample (Dow Chemical Co. data cited in Schwetz et al., 1978).

Other LD_{50} values for PCP following oral administration are 130 mg/kg for the mouse (Pleskova and Bencze, 1959), 168 mg/kg for the hamster (RTECS, 1977) and 250 mg/kg for guinea pigs (Gabrilovskaya and Laskina, 1964). The lowest reported oral lethal dose in man in 29 mg/kg (RTECS, 1977). Acute signs of intoxication are vomiting, fever, elevated blood pressure, increased respiratory rate, tachycardia and hyperglycemia (Knudsen et al., 1974).

Hoben et al. (1976) reported PCP more toxic in rats by the inhalation route than by ingestion. The LD_{50} for inhaled PCP aersol was 11.7 mg/kg in male rats. Exposure to 5 mg/l PCP dust for 1 hour, however, did not kill male and female rats (Reichhold Chemicals, 1974).

PCP may also be absorbed through skin. Dermal LD_{50} values of 96, 105 and 320 mg/kg have been reported for rats (Demidenko, 1966; Noakes and Sanderson, 1969; Gaines, 1969) and 261 mg/kg for mice (Pleskova and Bencze, 1959). Minimal irritation of intact and abraded skin have been observed in rabbits (Dow, 1969). Commercial but not purified PCP samples have produced chloroacne in the rabbit ear bioassay (Johnson et al., 1973); another rabbit ear bioassay test, however, indicated no discernable difference in results of exposure to various grades of PCP (Shelton, 1978). Rabbit eyes exposed to solid PCP showed slight conjunctival and iritic congestion (Dow, 1969).

With respect to the toxic effects of long-term exposure, only one chronic feeding study is available on PCP. Schwetz et al. (1978) fed weanling Sprague-Dawley rats 0, 1, 3, 10 or 30 mg/kg/day purified PCP in the diet for 22-24 months. The study of male rats was terminated after 22 months due to high mortality in both control and treated groups. Ingestion of 30 mg/kg/day of PCP produced a significant decrease in body weight in females; a significant increase in serum glutamic pyruvic transaminase activity in both male and female rats; an increase in the specific gravity of urine among female rats at the end of 1 year but not at 2 years; and an accumulation of pigment in the liver and kidneys. Pigment accumulation was also noted in females at the 10 mg/kg level. No other significant differences were observed. Tumor types and incidences were comparable in test and control groups.

Several subchronic feeding studies have also been reported. Kimbrough and Linder (1978) fed male and female Sherman rats 0, 20, 100 or 500 mg/kg of diet either technical grade or purified PCP for 8 months. None of the animals died during the study and no toxic manifestations were evident except a reduction in body weight gain in animals fed the highest mg/kg dose. Liver weights were significantly increased in rats fed 500 mg/kg of technical PCP. Morphologically, females were more severely affected than males. Livers of females were characterized by vacuolation of the hepatocytes, an increase in fibroblasts and other mononuclear cells within the sinusoids, bile duct proliferation, periportal fibrosis, degenerated liver cells, increased mitotic figures and an accumulation of brown pigments in macrophages and in Kupffer cells. Similar but less pronounced effects were observed in females at the 100 mg/kg level. The predominant lesion in males given 100 mg/kg and 500 mg/kg consisted of enlarged pleomorphic hepatocytes, which had foamy cytoplasm or cytoplasm with large vacuoles. Only mild alterations were noted in rats at the 20 mg/kg level.

In contrast, purified PCP caused slightly enlarged liver cells, with occasional eosinophilic cytoplasmic inclusions at 500 mg/kg but no alteration in the livers of rats fed the two lower concentrations. Rats receiving 500 mg/kg purified PCP also exhibited thickened walls in the hepatic central veins of the liver.

Johnson and co-workers (1973) also noted more severe toxic effects in rats fed 0, 3, 10 or 30 mg/kg/day of technical PCP than those observed in rats similarly treated with the purified material for 90 days. Technical PCP resulted in focal heptocellular degeneration and necrosis at 30 mg/kg and liver damage at the two lower treatment levels. No liver lesions were associated with treatment with purified PCP.

Similar findings in rats have been reported by Knudsen et al. (1974) Kimbrough and Linder (1975) and Goldstein (1977). Savolainen and Pekari (1979) reported that the administration of 20 mg/l of technical PCP in the drinking water of male Wistar rats for up to 14 weeks resulted in a significant liver concentration of PCP (56-65 μg/g wet tissue), which remained stable during the 14-week period. PCP also accumulated in perirenal fat (61 μg/g wet tissue at 14 wk) and brain (< 10 μg/g). The major portion of the PCP-body burden, however, was cleared within 4 weeks after switching to a PCP-free diet. Transient biochemical changes in brain enzymes were also noted during PCP treatment.

Goldstein (1977) recently examined the differences between the hepatic effects produced by commercial and purified PCP with respect to drug metabolizing enzymes and presence of porphyria. Female Sherman rats were fed pure of technical PCP at levels of 0, 20, 100 or 500 mg/kg of diet for

8 months. Technical PCP produced hepatic porphyria and increased hepatic aryl hydrocarbon hydroxylase activity, glucuronyl transferase activity, liver weight, cytochrome p-450, and microsomal heme, but no N-demethylase activity. The peak of the CO-difference spectrum of cytochrome p-450 was shifted to 448 nm, and there was a dramatic increase in the 455/430 ratios of the ethyl isocyanide difference spectrum. Enzyme changes were observed at 20 mg/kg of technical PCP; porphyria occurred at 100 and 500 mg/kg. Pure PCP had no significant effect on these parameters at any dose level except for an increase in glucuronyl transferase at 500 mg/kg. Both pure and technical PCP, however, decreased body weight gain comparably at 500 mg/kg. These results suggest that the liver changes associated with feeding technical grade PCP to rats stem from toxic contaminants rather than from PCP itself.

Thus, the toxicity of PCP cannot be readily divorced from the effects of contaminants in commercial formulations. Technical PCP is moderately toxic to mammalian species following acute ingestion or dermal exposure. Long-term dietary exposure of rats to 20 mg/kg or more of technical grade PCP is associated with liver lesions, porphyria and enhanced hepatic enzyme activity. Dietary levels up to 30 mg/kg/day of purified PCP, however, did not enhance the incidence of tumors in rats after 2 years.

4. Human Studies

Several cases of human poisoning with PCP or its sodium salt are cited in the scientific literature (Knudsen et al., 1974; Haley, 1977; Robson et al., 1969; Bevenue et al., 1967). PCP causes rapid uncoupling of oxidation and phosphorylation, cycles with a resulting increase in metabolic rate. Symptoms include fever, profuse sweating, dyspnea, congestion of ocular and nasal mucosae, abdominal pain, headache, fatigue and tachycardia (Young and Haley, 1978; Knudsen et al., 1974)

Uede et al. (1962) noted irritated throats, facial flushing and hand and leg weakness in four families after drinking and bathing in water from a well containing 12.5 mg/l PCP. Recovery occurred with 2-3 days. A similar case was reported by Chapman and Robson (1965) in a 4-year-old child who had bathed daily for 13 days with water from a PCP-contaminated holding tank. She was hospitalized with fever, intermittent delirium, acidosis, aminoaciduria and ketonuria but recovered completely.

Most cases of human exposure, however, are through direct dermal contact with PCP, from industrial exposure in wood treatment plants or inhalation of water vapor containing PCP in industrial cooling towers, paper pulp mills and tanneries.

Bevenue et al. (1967) reported reddening and development of painful sensations in the hands of a male 10 minutes after immersion of his hands in a solution containing 0.4% PCP. The pain persisted for 2 hours. One month after the episode, urinary PCP levels had returned to background levels.

Local skin irritation and chloracne have also been associated with dermal contact to PCP (Nomura, 1975; Knudsen et al., 1974). Johnson et al. (1973) however, demonstrated that the acnegenic agent was present in technical but not purified PCP, an observation suggesting dioxin contaminants as the causative agent.

A case of fatal aplastic anemia has been reported following dermal exposure to PCP for approximately one year (Roberts, 1963).

Begley et al. (1977) noted evidence of abnormal but reversible renal function in 18 workers at a wood treatment plant. Creatinine clearance and phosphorus reabsorption values were depressed prior to a 20-day vacation period but showed significant improvement during vacation, an occurrence suggesting that PCP exposure transiently reduced both the glomerular filtration rate and tubular function.

At least 24 industry-related PCP fatalities have been reported. At autopsy, the following changes are generally found: edematous brain, heart dilatation, tubular degeneration of the kidneys, edematous lungs with congestion and intra-alveolar hemorrhages, and a slightly congested liver with centrilobular degeneration (Knudsen et al., 1974).

5. Overview

Pentachlorophenol appears to be an ubiquitous contaminant of human tissues in industrialized societies; some 85% of human urine samples from the general population of the United States are positive for pentachlorophenol. PCP may be absorbed through the skin, by inhalation, or by ingestion of contaminated food or drink. The pharmacokinetics of PCP in man, however, show it to be readily excreted in urine, either unchanged or as the glucuronide conjugate.

Toxicological data on pentachlorophenol are complicated by the presence of varying amounts of polychlorinated dibenzo-p-dioxins and dibenzofurans in commercial formulations. Although pure PCP has not been fully characterized toxicologically, PCP does appear to be toxic in its own right. Acute LD_{50} values vary according to the contaminant content of the test material, but man would appear to be more susceptible than rodents and females more susceptible than males. Dietary exposure to 100-500 mg/kg technical PCP for 90 days is associated with pronounced liver lesions in rats; only mild alterations were seen at 20 mg/kg. Purified PCP produced no significant adverse effects in rats fed 3-10 mg/kg/day for up to 2 years.

No oncogenic effects were noted in either mice or rats fed 17 mg/kg/day and 30 mg/kg/day, respectively, of PCP for two years. Currently available data are insufficient to support the mutagenicity of PCP.

Reproductive effects, however, have been observed in rats orally exposed to 30 mg/kg/day or more during gestation. Purified PCP was somewhat more toxic than commercial grade PCP with respect to the incidences of resorbed fetuses, skeletal and soft-tissue anomalies and fetal growth retardation, particularly during early organogenesis. Comparable effects were not observed following a single but higher oral dose (60 mg/kg) of PCP.

B. HUMAN EXPOSURE

1. Introduction

The use of pentachlorophenol as a pesticide results in numerous opportunities for human exposure. As discussed previously in Part A of this chapter, PCP is commonly found in human urine, even in subjects who have not been occupationally exposed to the chemical. This has been taken by some as evidence of exposure, although the precise route or combination of routes is unknown. Detrick (1977) has suggested that PCP found in the body may be the result of the metabolism of other chlorinated compounds, such as hexachlorobenzene. This section discusses PCP exposure situations and quantifies the expected exposure to PCP whenever possible. It does not, however, consider occupational exposures such as those of pulp and paper mill workers or tannery workers. The exposure routes considered here are ingestion of food and drinking water, inhalation of air, and dermal absorption.

2. Ingestion of Food

Pentachlorophenol is not widely found in food. The FDA (1977), as part of the Total Diet Studies, reported PCP in 11 of 360 composite samples. Residues of 0.004-0.017 mg/kg were found in dairy products, grains and cereals, root vegetables, and sugars and adjuncts. On the basis of these data, the FDA estimated an average intake of 0.76 mg/day for a typical 15-20-year-old male. The USEPA (1978a) has calculated an average intake of 1.5 mg/day and a maximum of 18 mg/day based on average and maximum residues reported in the Total Diet Studies. However, these average intakes are based on the mean of the positive samples and are not actually representative of the average intake, since the actual incidence of PCP detection in food was low. As an example, PCP was detected in one composite sample of dairy products. However, in a smaller sample in Michigan, Lamparski _et al._, (1978) found no PCP in milk.

Various other food substances have been tested individually for PCP content. Section IV-B discussed levels of PCP in fish and showed a range of about 1-5 mg/kg wet weight. An average consumption of fish of 21.4 g/day (USDA, 1978) would result in an exposure of 0.1 mg/day, assuming a maximum level in fish of 5 mg/kg. Peanut butter has been indicated in the recent market basket study as a source of PCP in food. Heikes (1979) reported PCP in seven peanut butter samples at concentrations of 1.8-62 mg/kg, with an average of 18 mg/kg. The maximum sample was traced to a jar lid; however, most jar lids do not contain PCP. The author suggested that PCP may be a breakdown product of pentachloronitrobenzene.

which was also found in peanut butter. For an estimated daily consumption of 100 g of peanut butter, a maximum consumption of 6.2 mg/day PCP can be estimated. A more typical exposure from this source would be much lower, perhaps about 0.5 mg/day.

PCP has also been detected in gelatin. Firestone (1977) suggested that the chemical may have contaminated the hide used in making gelatin, since PCP is used as a preservative during hide processing. Levels of 0-8.3 mg/kg were found in 15 samples of gelatin, with higher levels found in that imported from Mexico. Such high concentrations of PCP in gelatin are expected to be rare so this exposure level represents a worst case. Using the maximum contamination of 8.3 mg/kg PCP in gelatin powder and a maximum consumption of 200 g of prepared jello per day, an ingestion of 24 μg/day can be estimated.

Another potential exposure route for humans is through ingesting food packaged in containers in which PCP was used as a preservative in glue. The propensity for different food types to absorb this PCP is unknown; however, the concentrations and total amount of PCP available in glues are probably small.

Thus, average intake of PCP in food has been estimated to be 1.5 mg/day. Persons consuming large amounts of peanut butter or gelatin would have potentially larger intakes, perhaps as much as 24 mg/day.

3. Ingestion of Drinking Water

PCP has been found in drinking water in the United States at low concentrations. EPA (1978b) sampled 113 finished waters in 1976 as part of the National Organics Monitoring Survey. PCP was found in 86 of 108 samples, with a mean of 0.07 μg/l and a maximum of 0.70 μg/l. However, the median was less than 0.01 μg/l, the minimum detectable limit. In a more recent survey supplementing the NOMS program, PCP was detected in eight cities out of 135 systems surveyed (U.S. EPA, 1980). Detected concentrations in finished water were somewhat higher than the NOMS results, ranging from 1.3 μg/l-12.0 μg/l. Assuming an intake of 2 l/day, exposure to most persons would be less than .02 μg/day, while the maximum would be 24 μg/day.

4. Inhalation of Air

Monitoring data for PCP in air is very limited. No information is available on ambient levels, and information on levels indoors is scarce. In pressure treatment plants and lumber mills using dip and spray treatment, Arsenault (1976) reported concentrations of 0.003-0.063 mg/m^3 PCP in areas where workers spent most of their time. Near the PCP source, however, levels ranged from 0.004 mg/m^3 to 1.000 mg/m^3. Assuming a breathing rate of 1.8 m^3/hr for 8 hours, exposure levels of 0.9 mg/day and 14 mg/day, respectively, can be calculated. These results are shown in Table 40. Though exposure in occupational settings is not considered in detail here, these data may provide an upper limit for exposure resulting from home use of PCP. The use of PCP in lumber yards in spray form is probably the most comparable to home use of PCP in terms of exposure; thus the maximum value of 0.063 mg/m^3 will be used. For an assumed breathing rate of 1.8 m^3/hr for 8 hours, this presents an exposure of 0.91 mg/day. However, a homeowner would most likely not be continually exposed to this level.

TABLE 40. SUMMARY OF LEVELS TO HUMAN EXPOSURE VIA VARIOUS PATHWAYS

Exposure Route	Exposure Level (mg/day)
Ingestion	
Average intake from food	0.76
Average intake from food (EPA)	1.5
Maximum intake from food (EPA)	18.0
Intake from fish (maximum)	0.1
Average intake from peanut butter	0.5
Maximum intake from peanut butter	6.2
Maximum intake from gelatin	0.024
Intake from drinking water (maximum)	0.024
Intake from drinking water (typical)	0.00002
Inhalation	
Working Subpopulation	
Wood treatment factions	
• general work area	0.9 (8 hrs)
• near PCP source	14 (8 hrs)
At cooling tower site	14 (8 hrs)
Use of PCP spray	0.9 (8 hrs)
General Population - national	
Evaporation of wood preserving wastewater	0.00009
Open burning of treated wood	0.0008
Evaporation from cooling towers	0.0007
Volatilization from treated wood	0.001
Total (summation of above)	0.0026
General Population - local	
Downwind of cooling tower (1 km)	2
Downwind of cooling tower (8 km)	0.06
Downwind of wood preserving wastewater evaporation pond (<1-4 km)	0.002
Inside house withPCP-treated wood room	3.2
Dermal Absorption	
Bathing and dishwashing with contaminated water	0.003 - 0.03
Home use of PCP as preservative	170
Handling of treated wood	0.5

Note: See text for assumptions.

Exposure levels for the general population can be estimated using the PCP concentrations in air calculated in Section IV-D. Nationally distributed upper limit ambient concentrations contributed from wood treatment wastewater evaporation, volatilization from treated wood, evaporation from cooling towers, and open-burning of wood, are 4.3 ng/m^3, 56 ng/m^3, 36 ng/m^3 and 40 ng/m^3, respectively. For an assumed breathing rate of 20 m^3/day, exposure levels of 0.09 μg/day, 1.1 μg/day, 0.7 μg/day, and 0.8 μg/day, respectively, can be calculated. These values, summed, give rise to a national exposure of 2.6 μg/day for the general population.

Likewise, the exposure of sub-populations downwind from cooling towers and wood preserving wastewater evaporation ponds can be estimated based on concentration estimates from Section IV-D. The upper limit plume concentrations estimated were 1.0 x 10^5 ng/m^3 (at a 1-km distance from a tower), and 100 ng/m^3 (up to -4 km distance from evaporation pond). Exposure levels calculated based on these concentrations are 2,000 μg/day, and 2 μg/day, respectively, assuming 20 m^3/day and a 24-hour exposure. If one assumes that the exposure within 1 km of a cooling tower applies only to an occupational subpopulation and that their exposure period equals 8 hours, then the resultant exposure level for this group is 14,400 μg/day. The USEPA (1978a) has estimated a daily exposure to 9,000 μg PCP for workers directly exposed to a cooling tower plume. It is unlikely, however, that humans would be directly exposed to the plume. The levels do indicate the magnitude of the potential for human exposure through inhalation of plume vapor and justify the need for further investigation of this pathway. Measurement of PCP concentrations in plumes would quickly verify the results of the concentration calculations.

While there have been no measurements of PCP in the air in the home, two reports in EPA's Pesticide Episode Review System (PERS) (U.S. EPA, 1976) may be relevant. One involved a painter exposed to "drift" while treating a roof. The other involved a man spraying PCP in a crawl space beneath a home. He developed weakness, headache, double vision, tachycardia, nausea and hyperpyrexia, and subsequently recovered. The route of exposure may have been dermal, as well as inhalation, though, the fact that the location was a crawl space suggests the latter. Furthermore the lack of dilution air volume may have resulted in a higher exposure than that estimated above.

Exposure of persons to PCP-treated wood, especially indoors, should also be considered. PERS (USEPA, 1976) reports several incidents of effects resulting from inhalation in treated rooms or houses. In some cases, the subjects had not applied the chemical so that inhalation was suggested as the likely route of exposure. The symptoms ranged from malaise to peripheral nerve manifestations. Gebefugl et al., (1976) measured air concentrations in enclosed areas treated with PCP according to the label directions in Germany. These authors found concentrations of 1-60 $\mu g/m^3$ PCP in the air. USEPA (1978a) used this maximum and a breathing rate of 1.0 m^3/hr. for 20 hours to estimate an exposure of 3.2 mg/day in this situation. Outdoor exposure to PCP-treated lumber would result in a substantially lower inhalation exposure.

5. Dermal Absorption

The estimation of exposure of persons through dermal absorption of PCP depends upon the assumptions used in the calculations. Unfortunately, since little is known about the permeability of the skin to different chemicals, only gross estimates of the exposure are possible. EPA (1978a) has used an estimate of 10% dermal absorption of PCP. However, numerous assumptions must be made in the use of this value, primarily in assuming the volume of the liquid in contact with the skin. The use of permeability constants may decrease the uncertainty. Therefore, in this section, a permeability constant of 0.01 cm/hour is used for PCP, similar to that which has been measured for phenol (Blank, 1979, personal communication). Though the validity of this assumption for PCP has not been tested, it does present a moderately high rate constant compared with those that have been measured, and thus represents a conservative approach to estimating exposure by dermal absorption.

The major sources of dermal exposure are occupational; the general public is exposed to low concentrations of PCP in water. Assuming daily contact (total body) with water for 0.2 hrs. through bathing and dishwashing, exposure levels of 0.0004 μg/day (at a mean PCP concentration of 0.01 ug/l) and 0.048 μg/day (at maximum PCP concentration of 12.0 μg/l) can be estimated.

Home use of PCP can also result in dermal exposure. The USEPA (1976) reported numerous incidents of perons spilling or splashing the solution on themselves. The symptoms observed were not usually described, but Bevenue et al. (1967) reported a burning sensation and skin irritation resulting from such an exposure. This type of exposure would not be chronic in most cases, however, since it is accidental, the extent of the exposure can vary widely. For the purposes of estimation, a 5% solution of PCP was assumed to have been spilled on 10% of the body surface area and to have remained there for 10 minutes. When the permeability constant of 0.01 cm/hour is assumed, the maximum estimated exposure is 170 mg.

The handling of treated wood may also result in PCP exposure. While persons such as carpenters, lumber yard workers, and loading dock workers handling crates would be exposed continually in this manner, homeowners would also be exposed occasionally. USEPA (1976) mentions a few incidents of this nature, without specifying the symptoms. USEPA (1978a) estimated the exposure to construction workers by assuming that 6 months after treatment, the concentration of PCP in the wood surface would be 0.5 mg/square foot. It was also assumed that the worker handled wood without gloves 40 times/day, and that the skin area exposed was 0.25 sq. ft., and that absorption was 10% effective. The resultant exposure was 0.5 mg/day.

6. Summary

Table 40 summarizes all the exposure levels calculated in the preceding section. The route of exposure to PCP for the largest number of people is through drinking water. While residues of PCP have been reported in food, the incidence appears to be low. The prevalence of high levels in gelatin is unknown. A typical exposure through drinking water was estimated to be 0.02 µg/day, and a maximum of 24 µg/day. The typical total dietary exposure was estimated at 1.5 mg/day and a maximum of 18 mg/day. However these estimates do not apply to as large a subpopulation as the drinking water estimates. Consumption of peanut butter in addition to other foods that contain maximum residues may contribute up to 24 mg/day for very limited subpopulations.

Other exposures considered here involve the use of PCP (herbicide or wood preservative) by the consumer or contact with PCP-treated wood. The maximum exposure through the use of PCP, as estimated here, appears to result from accidental spillage and dermal exposure. When various assumptions are made, the validity of which is unknown, an exposure of 170 mg/spill was estimated. Inhalation from treated wood indoors and during application may result in exposures of 1-3 mg/day. Inhalation of estimated ambient levels of PCP in the atmosphere (the summation of concentrations from four direct sources) has an associated exposure level of 6 µg/day for the general population. Subpopulations in the vicinity of 2.6 PCP sources may be exposed to significantly higher levels.

It should be pointed out that persons using PCP will be exposed through both the dermal and inhalation route. With appropriate precautions, the dermal route can be eliminated. However, it is not likely that inhalation exposure can be totally avoided.

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SECTION VI.

NON-HUMAN EFFECTS AND EXPOSURE

A. EFFECTS ON NON-HUMAN ORGANISMS

1. Introduction

This section provides information about the levels of pentachlorophenol that disrupt normal behavior and metabolic processes, as indicated by laboratory and field studies. The effects of the sodium salt of this compound, NaPCP, and of certain Dowicide chemicals containing PCP are also considered, as they are frequently used in bioassays because of their availability and solubility.

Several factors introduce a certain amount of inconsistency in bioassay results. The various grades of PCP range from less than 80% pure to 99+% pure, depending on the intended use of the material. Dowicides G and GS-T are pesticides (molluscicides) containing approximately 70-80% PCP, in addition to other chlorinated ingredients. These impurities may modify the toxicity of PCP, depending on their composition and relative concentrations (see Appendix A).

The type of bioassay used can also significantly influence the results. Tests conducted under static conditions are normally less reliable than continuous-flow experiments because there is less control of toxicant concentrations. However, continuous-flow conditions may not be suitable for testing certain kinds of organisms, such as larval fish and free-floating invertebrates. In both kinds of bioassays, the toxicant concentrations are often determined nominally (i.e., by diluting a measured amount of the substance) instead of by direct periodic measurement during the bioassay. Nominal determination of concentrations does not account for toxicant evaporation, adsorption onto particles or the test tank, or absorption by the test organisms, and so may produce significant overestimations of lethal and sublethal levels, particularly in static tests.

Water parameters, such as temperature, hardness and pH, have been shown to influence the toxicity of PCP to various aquatic organisms. Thus, variations in these parameters between experiments could yield conflicting or misleading results. Other factors include the species and developmental stage of the organism being tested. Since some species and stages may be more sensitive to PCP than others, it is not always valid to extrapolate the data from studies of related organisms.

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2. Freshwater Organisms

a. Chronic and Sublethal Effects

Low levels of toxicants that remain for extended periods are generally considered to represent "normally" polluted conditions in natural waterways. Under these circumstances, aquatic biota may become acclimated to the toxicant, or they may exhibit behavioral alterations such as increased respiration, loss of equilibrium, or reduced spawning and hatch success. Prolonged exposure, even to low concentrations of PCP, may ultimately result in mortality. Whether or not fish are killed by chronic exposure to PCP, certain effects, such as reduced spawning, may still endanger local populations.

Webb and Brett (1973) have studied underyearling sockeye salmon (Oncorhynchus nerka) in 12-week tests of growth rate and food conversion efficiency and 2-week tests of swimming performance. Growth rate and food conversion efficiency were closely related: the EC_{50} values (50% of organisms demonstrating the stated effect) were 1.74 µg/l and 1.80 µg/l respectively. Swimming performance, however, was unhindered at the highest exposure concentration (50.0 µg/l). Since the effects of these levels on natural populations are not known, they should not necessarily be considered sublethal or harmful.

Matida et al. (1970) found that the growth of rainbow trout (Salmo gairdneri) was "markedly retarded" after 4 weeks of exposure to PCP concentrations exceeding 8 µg/l, with slight retardation occurring at 3 µg/l. Solutions containing 34 µg/l of PCP created the same effect in sweetfish after 13 weeks of exposure. In the 2 µg/l solution, however, the experimental sweetfish grew more quickly than the control fish in 0 µg/l. The cause of this positive effect was not surmised by the authors; conceivably the low levels of PCP had a biocidal effect on the natural parasites and diseases of the sweetfish. Another seemingly paradoxical phenomenon was the variable effect of PCP on swawning by sweetfish. In 20 µg/l, spawning was delayed up to 2 weeks, while fish exposed to 34 µg/l spawned 7-10 days before the controls. Macroscopic inspection of ovaries and testes revealed no differences in fecundity and virility between experimental and test fish, and no explanation for the variation was given.

In acute toxicity tests, Reinert (1976) observed behavioral changes preceding death in rainbow trout. At concentrations between 10 µg/l and 32 µg/l, the trout exhibited abnormal respiration, followed by partial and total loss of equilibrium and death. Bluegill sunfish had the same symptoms in 65-100 µg/l PCP.

Chapman (1969) conducted a variety of tests on rainbow trout in various pre-adult stages. After 3-6 weeks of exposure to 30-100 µg/l NaPCP, trout alevins had retarded growth, increased yolk catabolism, and increased mortality compared with controls. The maximum dry weight of alevins reared in NaPCP decreased approximately 6% for each 10 µg/l increase in PCP concentration.

Whitley and Sikora (1970) observed an increase in respiration rates in sludgeworms (Tubifex tubifex) exposed to 250-1,250 μg/l PCP. This effect was attributed to the toxicant's tendency to interfere with oxidative phosphorylation. Respiration was affected to a greater degree at pH's exceeding 8.5, although the toxicity of the solution increased with decreasing pH.

Among the most sensitive freshwater organisms tested was the alga (Chlorella pyrenoidosa). Huang and Gloyna (1968) observed substantial destruction of chlorophyll at a concentration of 1 μg/l PCP; extrapolation of the data indicated that 7 μg/l would cause death. Blackman et al. (1954) exposed duckweed (Lemna minor) to PCP and determined an EC_{50} level (inducing chlorosis) of 800 μg/l. The minimum level reported to cause chlorosis was 121 μg/l.

b. Acute Effects

Acute toxicity is defined as toxicant-induced mortality over a short period, generally within 96 hours. Although fish in natural waterways are more likely to be exposed to lower concentrations that may result in chronic or sublethal effects, industrial discharges and spills can temporarily result in levels high enough to cause fish kills (see Section B of this chapter).

The acute effects of PCP on freshwater finfish have been studied for at least fifteen species, and this work provides a fairly reliable data base, which has been compiled and condensed in Table 41. It should be noted that the LC_{50} values given were derived under a variety of conditions. Such factors as exposure period (between 24 and 96 hours), age of test fish, differences in certain water parameters, and bioassay type (static or flow-through) may account for some of the variation in a given species' sensitivity. (Factors contributing to variability in PCP toxicity and fish sensitivity are discussed in greater detail in Part 5 of this section.)

Reported LC_{50} values for PCP and PCP salts range from 15.4 μg/l for the rainbow trout to 1,740 μg/l for the flagfish. As with many toxicants, the salmonids are apparently somewhat more sensitive as a group to PCP than other fish. However, the reliability of this generalization with respect to field situations is uncertain. Two normally hardy fish, the bluegill and the goldfish, were found to be relatively sensitive to NaPCP in a static bioassay by Inglis and Davis (1972).

LC_{50} values for the freshwater invertebrates tested ranged from 240 μg/l for the Oncomelania snail, to 10,000 μg/l for another snail species, Pseudosuccines collumnea. However, only four species are represented in the literature, and further testing could possibly reveal a broader range of sensitivities among the invertebrates. The available data are also summarized in Table 41.

TABLE 41. PCP ACUTE TOXICITIES (LC$_{50}$) TO FRESHWATER BIOTA*

PCP Concentration Range (ug/l)	Vertebrates
15.4[1] - 230[2]	Rainbow trout (*Salmo gairdneri*)
20[3] - 303	Bluegill sunfish (*Lepomis macrochirus*)
50[3] - 220	Goldfish (*Carassisu auratus*)
63[4] - 90[5]	Sockeye salmon (*Oncorhynchus nerka*)
64.2[5] - 150	Coho salmon (*Oncorhynchus kisutch*)
68 - 86	Sweetfish (*Plecoglossus altivelis*)
120	Channel catfish (*Ictalurus punctatus*)
130 - 135	Carp (*Cyprinus carpio*)
138	Brook trout (*Salvelinus fontinalis*)
160 - 170	Southern top-mouthed minnow (*Pseudorasbora parva*)
210 - 370	Fathead minnow (*Pimephales promelas*)
390	*Channa gachua*
510 - 630	Japanese killifish (*Oryzias latipes*)
1,000	Lamprey (*Petromyzon marinus*)
1,130[2]	Zebrafish (*Brachydanio rerio*)
1,740[2]	Flagfish (*Jordanella floridae*)
	Invertebrates
240 - 590[6]	Snail (*Oncomelania* sp.)
310 - 4,000	Sludgeworm (*Tubifex tubifex*)
680 - 4,000	Daphnid (*Daphnia magna*)
833 - 10,000	Snail (*Pseudosuccines collumnea*)

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[1] Reinert (1976)
[2] Fogels and Sprague (1977)
[3] Inglis and Davis (1972)
[4] Webb and Brett (1973)
[5] Davis and Hoos (1974)
[6] Hanumante and Kulkarni (1979)

3. Marine Organisms

a. Chronic Effects

The only marine finfish tested in a chronic exposure bioassay was the sheepshead minnow (Parrish et al., 1978). In continuous-flow conditions, adverse effects were observed in concentrations as low as 88 μg/l. Concentrations exceeding 88 μg/l caused significant mortality in adult fish during the 151-day test period. At 195 μg/l, hatching success and survival of second generation fish were also reduced.

The lowest concentration of PCP reported to cause sublethal effects in invertebrates was 40 μg/l, an EC_{50} level for abnormal development in the Eastern oyster (Borthwick and Schimmel, 1978). Rubinstein (1978) observed inhibition of feeding in the lugworm (Arenicola cristata) at at 80 μg/l.

The grass shrimp (Palaemonetes pugito) has been extensively studied for its reactions to various concentrations of PCP. Brannon and Conklin (1978) found that the exuviae (molted exoskeletons) of grass shrimp increased in dry weight with exposure to 100-1,000 μg/l PCP. It is possible, however, that this is an adaptive response rather than a sublethal effect. In a chronic bioassay, Conklin and Rao (1978) tested the sensitivity of shrimp to PCP during different stages of ecdysis, or shell molting. Shrimp in the later proecdysial stages were found to be more susceptible to PCP than during early proecdysial or intermolt periods. During 66 days of exposure to 436 μg/l PCP, 63% of the test shrimp died within 24 to 48 hours of ecdysis. Increased respiration rates were observed (as in sludgeworms) at a concentration of 5.0 μg/l by Cantelmo et al. (1978). Exposure to 10-20 mg/l caused an initial increase in oxygen consumption, which now was followed by a decline leading to death.

b. Acute Effects

Available data on acute toxicity to marine biota were somewhat limited, and covered only a small range of concentrations for finfish. The lowest reported LC_{50} for a vertebrate was 38 μg/l for the pinfish, by Borthwick and Schimmel (1978). The highest LC_{50} was 422 μg/l for the sheepshead minnow, as observed by Parrish et al. (1978).

The most sensitive invertebrate tested was apparently the Pacific oyster, with an LC_{50} of 48 μg/l. The highest LC_{50} recorded was for the shrimp (Palaemon elegans) in the adult stage at 10,390 μg/l. However, the same species also exhibited median mortality at 84 μg/l as first instar larvae in the same study. On the basis of such limited data for both vertebrates and invertebrates, it is not possible to identify particularly susceptible species, with the possible exception of the pinfish and the oyster. The data are summarized with references in Table 42.

TABLE 42. PCP ACUTE TOXICITIES (LC$_{50}$) TO MARINE BIOTA*

PCP Concentration Range (ug/l)	Vertebrates
38[1] - 53.2[2]	Pinfish (*Lagodon rhomboides*)
112[2] - 123	Striped mullet (*Mugil cephalus*)
240[1] - 422	Sheepshead minnow (*Cyprinodon variegatus*)
306	Longnose killifish (*Fundulus similis*)
	Invertebrates
48[3]	Pacific oyster (*Crassostrea gigas*)
84 - 10,390[4]	Shrimp (*Palaemon elegans*)
112 - 1,790[4]	Sand shrimp (*Crangon crangon*)
195	Brown shrimp (*Penaeus aztecus*)
250	Atlantic oyster (*Crassostrea virginica*)
363 - 5,090[4]	Shrimp (*Palaemonetes varians*)
436[5]- 3,650[6]	Grass shrimp (*Palaemonetes pugio*)

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*Data compiled from EPA (1978), except where noted.

[1] Borthwick and Schimmel (1978)
[2] Schimmel *et al*. (1978)
[3] Woelke (1972)
[4] van Dijk *et al*. (1977)
[5] Conklin and Rao (1978)
[6] Reinert (1976)

4. Other Studies

In an attempt to understand the impact of PCP on ecosystems, some researchers have devised methods to expose micro-communities to controlled levels of the toxicant. Tagatz et al. (1977, 1978) conducted tests on estuarine communities composed of annelids, arthropods, mollusks, and echinoderms. Ecocommunities were simulated by pumping seawater (with its plankton) into constant-head boxes with drains leading to a screened box, which collected animals from the effluent. PCP was metered at different concentrations into seawater flowing into each box.

In the first experiment, exposure concentrations were 7, 76, and 622 μg/l PCP for 9 weeks. At the lowest concentration, the only significant effect was a reduction in the number of mollusks compared with the control system. At 76 μg/l, annelids and arthropods decreased as well, while almost no animals survived at 622 μg/l.

In a second test, Dowicide G-ST (79% NaPCP) was used in concentrations of 1.8, 15.8, and 161 μg/l for 13 weeks. Although 1.8 μg/l had no effect, mollusks again were the most sensitive organisms in the macrobenthic community at higher concentrations, and the total number of benthic species declined.

Cantelmo and Rao (1978), using similar methods, found an increase in the biomass and density of nematodes in 76 μg/l as a result of the biocidal effects of PCP. However, concentrations of 161 μg/l and 622 μg/l were more harmful than beneficial. With increasing concentration there was a shift in relative abundance of espistrate feeders to deposit feeders. The general effects of PCP in all ecocommunity studies were reductions in the biomass and density of organisms, as well as changes in species composition. The latter effect could be the result of varying sensitivity between species, as well as alterations in the food supply caused by the algicidal properties of PCP.

5. Factors Affecting the Toxicity of PCP

There are several variables in a natural aquatic environment that may strongly influence the availability and toxicity of PCP to biota. Among the most important parameters are pH, water temperature and hardness. The relationship between fish size and sensitivity has also been studied, in addition to differential susceptibility to PCP in male and female snails. The interaction of PCP with other aqueous chemicals may modify its toxicity either by synergy or inhibition; however, such relationships remain to be studied in detail.

The pH of the water is perhaps the most significant and certainly the most studied of the parameters affecting PCP toxicity. As a weak acid, PCP dissociates only partially at a neutral pH (7.0). Even the dissociation of the sodium salt (most often used in bioassays) is pH dependent; at pH's above 7.4 it is 99% ionized (Davis and Hoos, 1974). Kaila and Saarikoski (1977) found that lowering the pH from 7.5 to

6.4 increased the toxicity of PCP to crayfish by a factor of 5.9, indicating that the nonionized form of PCP was more toxic, and by implication, more available for metabolism. The authors also suggested that the resistance of the crayfish may have been diminished by the acidic water.

Whitley (1968) found that the LC_{50} values for Tubifex worms doubled with each successive increase in pH between 7.5, 8.5, and 9.5. A study on fathead minnows by Crandall and Goodnight (1959) found similar effects of pH on PCP toxicity.

Whitley and Sikora (1970) observed two different effects of pH which occurred at opposite ends of the pH range. As described in Part 2 of this section, Tubifex worms exhibited significantly increased respiration rates at pH levels exceeding 8.5. The toxicity of PCP, however, increased with decreasing pH as in other experiments. The authors suggested that two separate mechanisms may be responsible for the two effects, and attributed the increased respiration response to PCP's effect on the uncoupling of phosphorylation. The mechanism for toxicosis was not suggested, although presumably the nonionized form of PCP affects metabolic systems differently.

Crandall and Goodnight (1959) also studied the effect of temperature on the toxicity of PCP to minnows. Survival times decreased significantly in response to a temperature increase from 10° to 26°C. The effect was attributed to the variable rates of metabolism as a result of the fish's poikilothermia. Asano et al. (1969) found that a 10°C rise in temperature reduced the median lethality period (LTM) and LC_{50} by factors of 2.23 and 4.82, respectively. In a bioassay with three species of marine shrimp, van Dijk et al. (1977) corroborated these findings using temperature variations of 5 to 25°C.

The data of Ruesink and Smith (1975), however, are completely at odds with these three studies. In 48- and 96-hour bioassays with fathead minnows, they found that PCP was more toxic by a factor of 1.5 to 1.8 as the temperature was reduced from 25° to 15°C. A suggested explanation was that the rate of detoxification and excretion may decrease more rapidly than the rate of absorption with decreasing temperature. Since no temperatures below 15°C were used in the test, however, these findings should be verified by further research.

Only one study testing the effects of water hardness on PCP toxicity was found. The data of Reinert (1976) indicate that rainbow trout and bluegill are significantly ($P = 0.05$) more susceptible in soft water (47 mg/l hardness) than in hardwater (218 mg/l). The respective 96-hour LC_{50}'s for trout and bluegill in hard water were 23 and 88 µg/l; in soft water, 15.4 and 79 µg/l. No explanation was offered for these results.

The size of the test fish may have some effect on its sensitivity, but experimental data are inconclusive. Matida et al. (1970) stated that the "median tolerance limit increases with the size of fish." Adelman et al. (1976) found that the size of goldfish had no significant effect on LC_{50} levels, while lethal threshold concentrations were slightly higher for smaller fish. This relationship was not evident in fathead minnows. In a bioassay with lake trout (Cristivomer namaycush), Goodnight (1942) concluded that "within reasonable limits the size of the fish...does not greatly affect the toxicity" of PCP or NaPCP. From the evidence presented, it appears that the relationship between size and sensitivity to PCP probably varies from species to species.

In an experiment conducted by Hosaka et al. (1969), Oncomelania snails were collected once a month for 7 months, with the males and females separated. Each group of snails was then exposed to several concentrations of NaPCP for 48 hours. By the fifth month females were more resistant than males, while the LC_{50}'s were the same in one group of snails collected in June (the sixth month). The females particularly became more sensitive between April and June, the time coincident with an egg-laying period. Sexual and reproductive factors remain to be investigated for other species.

The presence of other chemical substances in the water may increase or decrease the toxicity of PCP. The only substances tested so far have been carbaryl and arecoline, a direct-acting cholinergic agent (Statham and Lech, 1975). The toxicity of pentachlorophenol to rainbow trout was significantly increased in fish that were pre-exposed to these compounds, even though carbaryl by itself caused no mortality during 48 hours of exposure to a 1 mg/l concentration.

Various bioassays on PCP toxicity have used different grades of the toxicant, which could be a significant factor in LC_{50} variability. Technical grades of PCP may contain less than 80% of PCP; commercial grades a higher proportion; and reagent grades in excess of 99.8% PCP. Depending on the toxicity of these impurities, experimental results under otherwise identical conditions can vary widely. For a discussion of the contaminants found in PCP, see Appendix A.

6. Effects on Terrestrial Biota

a. Plants

The only study available on PCP toxicity to land plants was done by Sund and Nomura (1963). The respective LC_{50} values for radish (Raphanus sativus) seeds and grass (Sorghum sudanense) seeds were 7.190 and 5,400 μg/kg. Blackman et al. (1954) observed inhibition of radial growth in the mycelium of the fungus, Trichoderma viride, at 585 μg/kg.

b. Animals

No data on PCP effects on terrestrial animals were available, aside from scattered information in Vermeer et al. (1974). See Section VI-B for a discussion of this study.

7. Conclusions

According to the literature surveyed, the lowest concentration of PCP at which effects have been observed in an aquatic organism is 1 μg/l, which caused destruction of chlorophyll in the alga, Chlorella pyrenoidosa. The sockeye salmon exhibited reduced growth and food conversion efficiency at 1.74 and 1.80 μg/l, respectively. The lowest reported LC_{50} is 15.4 μg/l, for the rainbow trout. Oncomelania snails were apparently the most sensitive freshwater invertebrate tested, for which the LC_{50} values ranged from 240 μg/l to 590 μg/l.

Among the marine organisms, the pinfish and the Pacific oyster were the most susceptible to PCP, with respective LC_{50}'s of 38 μg/l and 48 μg/l. Three species of marine shrimp were found to be substantially more sensitive during the larval stages than as adults.

PCP has been shown to affect estuarine ecosystems adversely by reducing biomass and species diversity and composition.

The toxicity of PCP is affected by several water parameters. The data generally indicate that toxicity increases with decreasing pH, increasing temperature, and decreasing hardness. Factors of uncertain importance are fish size and interactions with other aqueous chemicals. The study of PCP toxicity could be expedited by the adoption of a standard grade of PCP for use in aquatic bioassays, as purities of different grades range from less than 80% to 99+% PCP.

B. NON-HUMAN EXPOSURE

1. Introduction

Fish kill data indicate that PCP sometimes reaches concentrations in natural systems high enough to have lethal effects on aquatic organisms. Unfortunately, monitoring data reporting ambient PCP levels in surface water are too few to support more than speculation on exposure of aquatic biota to PCP.

2. Monitoring Data

The data provided by STORET on PCP levels in ambient waters of U.S. river basins are extremely limited, indicating that PCP is an infrequently monitored parameter. The California and Pacific Northwest basins (with 41 and 22 measurements, respectively) are the only areas with even a remotely adequate data base. Remarked and unremarked measurements ranged from 1 ng/l to 100 mg/l, with the majority of values between 1 μg/l and 100 μg/l. Levels in California were fairly evenly distributed between 10 ng/l and 10 μg/l. The Pacific Northwest had substantially higher values than California, with 91% of the data in the 10-100 μg/l range.

Ambient concentrations in surface water from sources other than the STORET system are also few and inconclusive. Because these data are commonly accompanied by a description of the location (e.g., near wood preserving plant), source of the contamination (e.g., run-off following storm), they provide examples of concentrations that fish may be exposed to in the field. The highest reported PCP concentrations were measured in an industrial area and ranged from 82 µg/l to 10,500 µg/l (see Table 6). A higher concentration of 5450 mg/l was found in an oil sample at a factory outlet (Table 6). Most of these miscellaneous observations, however, were consistent with the concentrations reported in STORET, usually less than 100 µg/l.

3. EXAMS Model

The EXAMS model (U.S. EPA 1980) was used to simulate concentrations of PCP in natural aquatic systems based on realistic loading rates. The results are discussed in detail in Section IV-D-2 (see Tables 34-37). The maximum values generated by the model for water concentrations are summarized in Table 43. Most of the data are fairly consistent with measured ambient concentrations reported in STORET and elsewhere: less than 100 µg/l. At the highest loading, 17.1 kg/day, concentrations are higher than typical measured concentrations, especially in the pond and eutrophic lake systems. The highest measured concentration reported, 10,500 µg/l, was greater than the highest EXAMS value.

The results of the EXAMS model estimate the significance of build-up of PCP concentrations based on reported dishcarge rates. Due to data limitations and the level of effort that would be required, the generalized aquatic systems models in EXAMS were used without adjustment that might more accurately simulate aquatic systems into which PCP is actually discharged. System influences on concentrations were very noticeable. Maximum estimated concentrations for rivers were lower than those for static systems, the small pond system achieved the highest concentrations, and the eutrophic lake had higher levels than the oligotrophic lake. Bottom sediment concentrations were approximately three to fourteen times the water concentrations.

If the EXAMS model is assumed to simulate reasonably the concentrations accumulating in different aquatic systems during continuous PCP discharge, then the results can be used to gain further insight into the impact of PCP effluents on aquatic systems in general. The simulated concentrations do not, however, represent the worst case because the model assumes reduction of pollutant concentrations at the transformation rates entered as input data as well as loss through the physical transport mechanisms appropriate to each system.

4. Fish Kill Data

Table 44 provides information on the location and activities associated with fish kills attributed to PCP between 1970 and 1976. Unfortunately, no data on PCP concentrations were available in the fish kill

TABLE 43. WATER CONCENTRATIONS ESTIMATED BY THE EXAMS MODEL FOR DIFFERENT PCP LOADING RATES (μg/l)

	Concentration (μg/l)					
PCP Loading (kg/day)	Pond	Eutrophic Lake	Oligotrophic Lake	River	Turbid River	Coastal Plain River
0.07	107.9 (87.5)[1]	0.1 (0.079)	0.005 (0.002)	0.003 (0.002)	0.003 (0.002)	.032 (.028)
0.25	385.4 (312.5)	3.33 (1.75)	0.016 (0.008)	0.011 (0.010)	0.011 (0.010)	0.115 (0.101)
17.1	26362 (21375)	227.9 (121.2)	1.14 (0.584)	0.784 (0.704)	0.784 (0.704)	7.84 (6.91)

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[1] First number is total PCP concentration, number in parenthesis is dissolved PCP concentration).

TABLE 44. DATA FOR PCP-RELATED FISH KILLS, 1970-1976

Year	River	Nearest City	No. Killed[1]	Cause
1970	Cahaba R.	Centreville, AL	65	Lumber treatment
1970	Caney Cr.	Conroe, TX	30	Railroad pilings
1971	Saquoit Cr.	Clayville, NY	3	Paper plant slime control
1972	-	PA	3	Possible contamination from mushroom growers
1972	-	OR	563	Runoff from lumber company
1972	Big Piney R.	Cabool, MO	115	Chemical industry operations
1972	Shipbuilder's Cr.	Webster, NY	10	Publishing Company
1973	Mill Cr. & tribs	Webster, NY	1	Cooling water from copying machines
1973	Mill Cr. & tribs.	Webster, NY	2	Cooling water from copying machines
1974	Brickyard Cr.	Red Bluff, CA	.018	Cans thrown into creek
1974	-	CA	7	-
1974	Napa R.	Calistoga, CA	3.1	PCP spraying on bridge
1974	San Leandro Cr.	San Leandro, CA	1.6	PCP spraying on bridge
1974	Dryden L. outlet	Dryden, NY	1	-
1975	-	MO	?	Runoff from power plant cooling tower
1976	Anderson Cr.	Anderson, CA	1	Paper industry
1976	-	GA	9	-
1976	-	AR	2	Discharge from pallet company
1976	Cedar Cr.	Cedartown, GA	0.2	Rail operation - preservative spill
1976	Cowskin Cr.	Wichita, KS	25	Chemical operations
1976	Mtn. Fork R.	Mena, AR	8	Paper industry
1976	Bear Cr. Res.	Bear Creek, AL	38	Wood preservative discharged from holding lagoon at lumber lumber company
1977	Port of Stockton	Stockton, CA	1	PCP discharge from co. treating logs with creosate
1977	Texaco Canal & Crawford Canal	Paradis, LA	unknown	NaPCP from petroleum operation.

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[1] In thousands.

Source: United States Environmental Protection Agency (USEPA) 1979a. Reports of fish kills, 1970-1976. Data files of Monitoring and Data Support Division reports; moreover, the presence of other toxic chemicals cannot be ruled out. It is possible that, in some cases, synergy between PCP and other toxicants increased the magnitude of the fish kill beyond the effect of PCP alone. Lumber and wood product industries were the most frequent sources of spills (from holding lagoon overflows), discharges, and runoff from plant sites. During use as a wood preservative near water, PCP also caused fish kills. Another source of PCP was discharge of cooling water used in power plants and copying machines. With regard to geographical distribution, the reported fish kills were not concentrated in any one region of the country.

5. Exposure to Terrestrial Animals

All of the information on PCP exposure to land animals were taken from a field study by Vermeer et al. (1974) in rice field of Surinam, South America. The study area was an 8000-hectare rice-growing project, where some dozen pesticides were used heavily. Applications of NaPCP were the heaviest, at a volume of 50,000 kg per year. The concentration of NaPCP in the rice fields averaged about 4 mg/kg following application. Among the terrestrial animals found at the study site were 33 species of birds, a frog (Pseudis paradoxa), and the spectacled caiman. Six times as many dead snail kites (Rostrhamus sociabilis) were found at a roost close to the rice fields as in a roost further away. The most likely exposure route was the ingestion of contaminated Pomacea snails. Dead specimens of egret, heron, and josana were also found, but there was no basis for comparison with mortality rates in unaffected birds.

PCP is normally not used directly on crops or in rice fields on a large scale in the U.S. Application rates for use as a garden herbicide are lower and the area typically treated is not as large as an agricultural field. Thus exposure incidents such as the one previously described are unlikely to occur in the U.S. This does not rule out small-scale exposure of local invertebrate and bird populations to PCP used as a garden herbicide. In addition, there may be exposure to PCP spilled on soil and plants surrounding wood treatment plants. Another pathway is ingestion of contaminated fish. Accumulation of PCP up to three orders of magnitude (depending on the species) above water concentration has been reported (see Section IV-C).

6. Summary

Aquatic organisms are typically exposed to PCP in water at concentrations less than 100 µg/l. Simulations of water concentrations resulting from practiced discharge rates show agreement with these exposure levels, with the exception of discharges to the pond system and at the highest loading rate to the eutrophic lake system. Higher water concentrations have been measured in industrial areas, the highest at 10,500 µg/l. Even higher concentrations may be found associated with oil slicks on water surfaces near sources. Incidences of fish exposure to PCP have been reported in the vicinity of wood preserving plants, pulp and paper industries, cooling water discharges, and following spraying.

Terrestrial organisms may be exposed to PCP through contact with spilled material and ingestion of contaminated plants and aquatic organisms. No information regarding actual exposure was available.

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SECTION VII.

RISK CONSIDERATIONS

A. METHODOLOGY

The desired output of the risk assessment is the quantification of risks to various subpopulations of humans and other classes of biota. This requires careful identification of the subpopulations at risk and the populations exposed, evaluation of the ranges in each subpopulation's exposure consideration of the effects levels or dose response data for the species of concern and/or proxies for these species and extrapolation of effect levels from dose/response data for laboratory animals to the human subpopulations at risk.

In this risk assessment for PCP, exposure levels that might result from several human exposure scenarios are compared with levels of PCP exposure that have caused effects in laboratory animals. For a number of reasons, no attempt has been made to extrapolate dose-response relationships for humans. The risks most clearly associated with exposure to PCP are the reported teratogenic and reproductive effects. At present, there appears to be no generally accepted basis for extrapolation of teratogenesis data from laboratory animals to humans. As a result, the discussion or risk for humans is qualitative only. For the exposure scenarios, possible exposures of humans derived from a number of scenarios, and the "safety factors" are computed from the ratio of effects to exposure levels. These provide an indication of the significance of PCP as a potential environmental hazard to humans.

In the case of risks to other forms of biota, insufficient data are available on most toxic effects and on exposure values to permit quantitative risk estimates. However, potential risks to various species are described and presented herein.

B. RELATIVE EXPOSURE EFFECTS AND RISK COMPARISONS--HUMANS

Table 45 presents a series of possible non-occupational exposure scenarios for humans, with an indication of the possible exposure routes and levels and key assumptions used in developing the exposure estimates. Data used in developing the estimates in this table were described in other sections of this report. The size of the subpopulation associated with these exposures is described qualitatively and is only meant to indicate the relative distribution of each exposure scenario in the population.

The maximum exposure to PCP in food would 0.4 mg/kg/day. Contributions from drinking water, even for the maximum contamination level, are relatively small. It should be noted, however that residues in food are not commonly found in sampling studies. Therefore, the maximum possible exposure is probably only valid for a very small subpopulation.

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TABLE 45. EXPOSURE OF HUMANS TO PCP

Exposure Route and Medium		Exposure (mg/day)	Exposure mg/kg /day[1]	Subpopulation	Assumptions[2]
Ingestion					Typical 5-20 yr male, average residues.
Food					
Diet - "Typical"		1.5[3]	0.025	small	Typical 15-20 yr. male, maximum residues
	Maximum	18[3]	0.3	very small	Typical 15-20 yr. male, maximum residues
Fish -	Maximum	0.1	0.002	very small	Maximum concentration in fish of 5 mg/kg and an average consumption of 21.4 g/day.
Peanut butter					
	"Typical"	0.5	0.008	probably small	Concentration of 5 mg/kg, consumption 100 g/day
	Maximum	6.2	0.1	very small	Concentration of 62 mg/kg, consumption 100 g/day
Gelatin	Maximum	0.024	0.0004	probably small	Concentration of 8.3 mg/kg in gelatin; consumption 200 jello/jelly/day
Total	Maximum	24	0.4	very small	
Drinking Water					
	Typical	0.00002	-	large	Concentration of 0.01 mg/l, consumption - 2 l/day.
	Maximum	0.024	0.0004	very small	Concentrattion of 0.01 mg/l, consumption - 2 l/day.

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[1] For a 60-kg person (woman).

[2] See Section V for more detail.

[3] Includes drinking water

TABLE 45. EXPOSURE OF HUMANS TO PCP
(Continued)

Exposure Route and Medium	Exposure (mg/day)[1]	Exposure mg/kg /day[1]	Subpopulation	Assumptions[2]
Inhalation				
Downwind of cooling tower (1 km)	2	0.03	very small	Upper limit concentration of 100 μg/m^3, inhalation, 20 m^3/day.
Downwind of cooling tower (8 km)	0.06	0.001	small	Upper limit concentration of 3 μg/m^3, inhalation of 20 m^3/day
Home use of PCP-Maximum	1	0.02	small	Concentration of 0.07 mg/m^3, breathing rate - 1.8 m^3/hr, exposure time - 8 hrs.
Air in PCP treated rooms	3.2	0.05	very small	Concentration of 0.16 mg/m^3, breathing rate - 1.0 m^3/hr, exposure time - 20 hrs
Outdoors-general population	0.003	0.00005	large	See Section V
Dermal Bathing, dishwashing				
Typical	0.0000004	-	large	Concentration of 0.01 μg/l, permeability constant 0.01 cm/hr, total body immersion - 0.2 hr/day.
Maximum	0.00048	-	small	Concentration of 12.0 μg/l permeability constant 0.01 cm/hr, total body immersion - 0.2 hr/day.
Home use-spill maximum	170	2.8	very small	Concentration of 50,000 mg/l, permeability of 10% body surface area, for 10 minutes/spill.
Handling of treated wood -	0.5	0.008	small	Concentration of 0.5 mg/ft at wood surface, absorption 10%, 40 contacts of 0.25 sq. ft/contact

Exposure through normal inhalation or dermal absorption also appears to be quite low. However, certain situations present the potential for considerably higher exposures, primarily use-related activities. The home use of PCP can result in an estimated maximum exposure of 0.02 mg/kg/8 hrs. In addition, spills of PCP may result in direct dermal exposure, perhaps as much as 170 mg, as well as inhalation exposure. However, these exposures would not be recurring ones.

Other use-related situations can result in chronic exposure. For example, the use of PCP-treated wood indoors may result in an inhalation exposure of 0.05 mg/kg/day. The continued use of PCP in these situations would result in a chronic exposure. In addition, persons living close to cooling towers may be exposed to maximum levels of 0.03 mg/kg/day.

Table 46 combines the estimates shown in Table 45 into daily total doses for three scenarios. In the worst case situation, food can be the largest contributor to exposure on a chronic basis. This is also true under more typical circumstances. However, it should be reiterated that the magnitude of the subpopulation experiencing these exposures is not known. For example, the exposure of persons to 0.00002 mg/day in drinking water is probably much more typical than exposure to 1.5 mg/day in food. In the case of persons living close to cooling towers, inhalation may represent the more significant exposure pathway.

Table 47 indicates levels of exposure to PCP at which adverse effects have been observed in laboratory animals. (These data are discussed in more detail in Section V.) Fetotoxic and teratogenic effects have been observed in the rat following exposure to 15-30 mg/kg. This exposure is critical primarily during specific phases of gestation, and does not necessarily have to be a chronic exposure to result in teratogenic effects. With the apparent effect level of 6 mg/kg for these effects, the safety factor for the exposure estimates can be evaluated.

The worst case situation described in Table 46 would result in an exposure to 3.2 mg/kg/day, providing a safety factor of 1.7 over the reported effects level (6 mg/kg). However, this number exceeds the OPTS no effects level of 3.0 mg/kg/day (U.S EPA 1980). Most of the exposure in this scenario can be attributed to a spill of PCP. Thus, such an incident would be of concern during pregnancy. When the spill is excluded, the exposure in this case would be only 0.4 mg/kg, associated with high residues in food. The safety factor in this case would be 8.6. Though it is likely that these situation are extremely rare, the exposure routes mentioned above should be examined more closely in order to determine actual exposures and the size of the subpopulations associated with each.

Other non-occupational exposure routes provide a safety factor of at least 100. This is not to say, however, that they represent acceptable levels of exposure since there are considerable uncertainties in all aspects of such an assessment.

TABLE 46. EXPOSURE SITUATIONS FOR PCP

Exposure Situation/Pathway	Exposure (mg/day)	Exposure (mg/kg/day)
Maximum Exposure		
Food	24	0.4
Drinking Water	0.024	0.0004
Inhalation - Ambient	0.003	--
Dermal - Home Use - Spill	170	2.8
TOTAL	194.0	3.2
Exposure of Typical Person[1]		
Food	1.5	0.025
Drinking Water	0.00002	-
Inhalation Ambient	0.003	-
Dermal	0.003	-
TOTAL	1.5	0.025
Exposure of Person Living Near Cooling Tower		
Food	1.5	0.025
Drinking Water	0.00002	-
Inhalation	2	0.03
Dermal	0.003	-
TOTAL	3.5	0.06

[1] It is not known how "typical" these exposures are; the levels in drinking water are known to be low and numerous locations have been sampled. No monitoring data is available for air. Limited data are available for food, and the detection of PCP was not widespread.

TABLE 47. REPORTED LEVELS OF PCP CAUSING ADVERSE EFFECTS IN MAMMALS

Adverse Effect	Species	Lowest Reported Effect Level	No Apparent Effect Level
Carcinogenesis	Rat	- - -	30 mg/kg/day (diet)
	Mouse	- - -	17 mg/kg/day (diet)
Fetotoxicity	Rat	15 mg/kg	5.8 mg/kg[1]
Teratogenesis	Rat	30 mg/kg (days 8-11)	- - -
Liver Lesions	Rat	100 ppm (diet)	- - -
Median Oral Lethal Dose	Rat	78-205 mg/kg	- - -
Median Inhalation Lethal Dose	Rat	11.7 mg/kg	- - -
Median Dermal Lethal Dose	Rat	96-320 mg/kg	- - -

[1] OPTS has decided on 3 mg/kg/day as a no effects level for fetotoxic effects in rats (U.S.EPA 1980).

The above discussion has dealt with general human exposure scenarios and reported teratogenic effect levels for laboratory animals. There is also some very limited epidemiological evidence of a link between PCP exposure via treated wood and leukemia. In a study in Kentucky, 140 workers handling packing cases treated with PCP (average concentrations of 127.4 μg/kg), appear to have an abnormal incidence of leukemia (Dickson, 1980, Pesticide and Toxic Chemical News, 1980). Since the dioxin contaminants have also been implicated, the evidence regarding PCP is unclear. The results of this study should become available in the near future and should enable a more thorough consideration of this possible risk of PCP exposure.

C. RELATIVE EXPOSURE EFFECTS AND RISK COMPARISONS--BIOTA

The monitoring data provided by STORET and other sources were insufficient to permit estimation of the potential for harmful exposure of aquatic organisms to pentachlorophenol on a national level. Measured concentrations in major river basins ranged from 1 ng/l to 100 μg/l. Data were heavily skewed by the preponderance of observations from California and the Pacific Northwest. Levels in California were fairly evenly distribured between 10 ng/l and 10 μg/l, while 91% of the measurements in the Pacific Northwest were in the 10 μg/l to 100 μg/l range. It is worth noting that both river basins are locations of wood preserving industries.

Simulation of water concentrations generated for known PCP discharge rates through use of the EXAMS model yielded levels (of total PCP) ranging from 3.2. x 10^{-6} mg/l (at a PCP discharge of 0.07 kg/day) in a riyer system to 26.36 mg/l (at a PCP discharge of 17.1 kg/day) in a pond system. Most concentrations for all systems excluding the pond were less than 1.0 mg/l. Again, it should be noted that these concentrations are based on numerous assumptions described in Sectin IV-D-2, including the unconservative assumption that PCP leaves the system at the rates entered as input to the model. Not all transfer and transformation reactions can be expected to take place at all sites of PCP discharge. At the two highest loadings for the pond and lake systems, 17.1 kg/day and 0.25 kg/day, the simulated total PCP concentrations violated or were only slightly less than the freshwater criterion level for PCP of 14 μg/l recommended by EPA (USEPA, 1978). At the lowest loading of 0.07 kg/day, the estimated concentration of PCP exceeded the freshwater criterion in the pond system and was slightly below it in the oligotrophic lake system. In the river systems, none of the estimated concentrations of PCP exceeded the criterion level, even at the highest loading; however, in the coastal plan river system the estimated concentration was only slightly under the freshwater criterion.

From the toxicity data, it appears that PCP concentrations greater than 1 μg/l may cause chronic or sublethal effects in aquatic biota. Levels exceeding 10 μg/l could be lethal to many species over varying periods of exposure.

One species of alga, Chlorella pyrenoidosa, has been adversely affected by PCP concentrations as low as 1 μg/l. Rainbow trout were the most susceptible of the freshwater fish, with a 96-hour LC_{50} of 15.4 μg/l; other salmonids were found to be comparatively sensitive as well. Among the marine organisms the pinfish and the Pacific oyster were the most susceptible to PCP, with respective LC_{50}'s of 38 and 48 μg/l. Most fish were sensitive in the ranges of 10-300 μg/l. In most aquatic animals, larval and juvenile stages are periods of decreased resistance to most toxicants.

Among the factors that influence PCP toxicity, pH is perhaps the most significant. Since PCP is more toxic in its nonionized form, a lower pH increases risk to biota. From the limited evidence, it appears also that higher temperature and softer water increase PCP toxicity; however, these effects remain to be verified by further research. Where PCP occurs in the water, fish will generally be more susceptible in warmer, softer, and more acidic waters.

When environmental variables (e.g., pH, hardness) that may modify PCP's toxicity in natural ecosystems are not considered and on the basis of a very limited monitoring data base, certain freshwater fish and invertebrate species appear likely to encounter PCP at concentrations to which they are sensitive, greater than 1.0 μg/l. According to the EXAMS model, the currently practiced discharge rates of 0.25 kg/day and 17.1 kg/day (see Table 33) may result in water concentrations on the order of 0.1 to 26 mg/l, especially in the more static aquatie systems such as ponds and lakes. Lower practiced discharge rates do not appear as likely according to the model, to generate concentrations at levels harmful to biota except in small ponds. Discharges are assumed less likely to be released into ponds than into larger, more resilient aquatic systems. Exceptions may be contained ponds that are considered as holding lagoons; however, these are not natural systems and the EXAMS model would not be applicable to them. It should be pointed out again that the estimated loading rates are based on small samples of effluent concentrations and flow rates represent neither the highest known rates of PCP discharge into water, nor typical discharge practices.

Fish kill data and miscellaneous observations of high PCP concentratations in water implicate several industries and activities that may generate levels of PCP potentially harmful to aquatic life. These include the wood preserving industry (through holding lagoon spills, runoff from contaminated soil), the pulp and paper industry, cooling water discharges and spraying of wood near water.

Individual continuous industrial discharges on the order of 0.25 kg/day and 17.1 kg/day may result in harmful concentrations in certain aquatic systems. No information on effluent levels or discharge practices was available for a number of PCP-associated industries. In addition, since currently available effluent data are based on a limited number of observations, they may not reflect widespread discharge practices. Further investigation is warranted on disposal practices for cooling waters from cooling towers and machinery, in particular.

The PCP contribution to water from treated wood and from boat paints treated with PCP also requires further investigation. Treated wood distribution is widespread throughout the U.S. and an estimated 18,000 MT of PCP is consumed annually by the wood preserving industry alone, although the percentage of the total mass coming in contact with water is unknown. There is a potential for leaching out of the chemical from wood submerged in water (as dock posts or on boat bottoms); however, no fate data were available to permit estimation of the significance of this process.

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APPENDIX A. CONTAMINANTS IN PENTACHLOROPHENOL

The contaminants in commercial pentachlorophenol could play a role in the environmental importance of PCP. Commercial technical grade pentachlorophenol contains contaminants, mostly other chlorinated phenols, dioxins, and dibenzofurans (see Figure A-1).* The chlorinated phenols include 2,3,4,6, tetrachlorophenol, traces of trichlorophenol, chlorophenoxy phenols, chlorodiphenyl ethers, chlorohydroxy diphenyl ethers, and traces of even more complex phenol reaction products. The principal chlorodioxins and chlorodibenzofurans are those containing six to eight chlorine atoms. The highly toxic 2,3,7,8 tetrachlorodibenzo-p-dioxin (TCDD) isomer has not been identified as a contaminant of PCP manufactured in the U.S.

Octachlorodibenzodioxin (OCDD) is the principal dioxin contaminant. There are two heptachlorodibenzodioxins and both have been found in PCP. Of the ten possible hexachlorodibenzo-p-dioxin isomers possible, six have been detected. (See Table A-1 for a listing.)

Several dibenzofurans have also been detected in PCP. These include tetrachlorodibenzofurans, pentachlorodibenzofurans, and a hexachlorodibenzofuran (see Table A-2).

The relative concentrations of these contaminants in PCP are shown in Tables A-3 and A-4. The composition of PCP from various sources varies somewhat.

Commercial processes exist that enable the production of PCP with greatly reduced concentrations of contaminants. However, the production of a purified product is more costly, and may result in byproducts that are more difficult to handle. Disposal of the highly concentrated residual waste that contains the removed contaminants is problematic. For every 5000 units of PCP manufactured, approximately 10% of this amount, or 500 units, of bottom residues are produced; these may contain the following compounds at varying concentrations: octa-, hepta-, and hexa-chlorodibenzodioxins and chlorodibenzofurans, tetrachlorophenol, trichlorophenol, chlorophenoxy phenols, hydroxy diphenyl ethers, hexachlorobenzene, chlorinated biphenyls, chlorinated polymers, and other compounds. These figures indicate that approximately 2000 tons/year of residual wastes requiring disposal are generated.

The removal of contaminants during production may not be as complete as one might expect. Many of these contaminants are also degradation products of PCP and can be expected to be formed in the environment. Therefore, the initial removal of these contaminants during production does not assure that they will not enter the environment.

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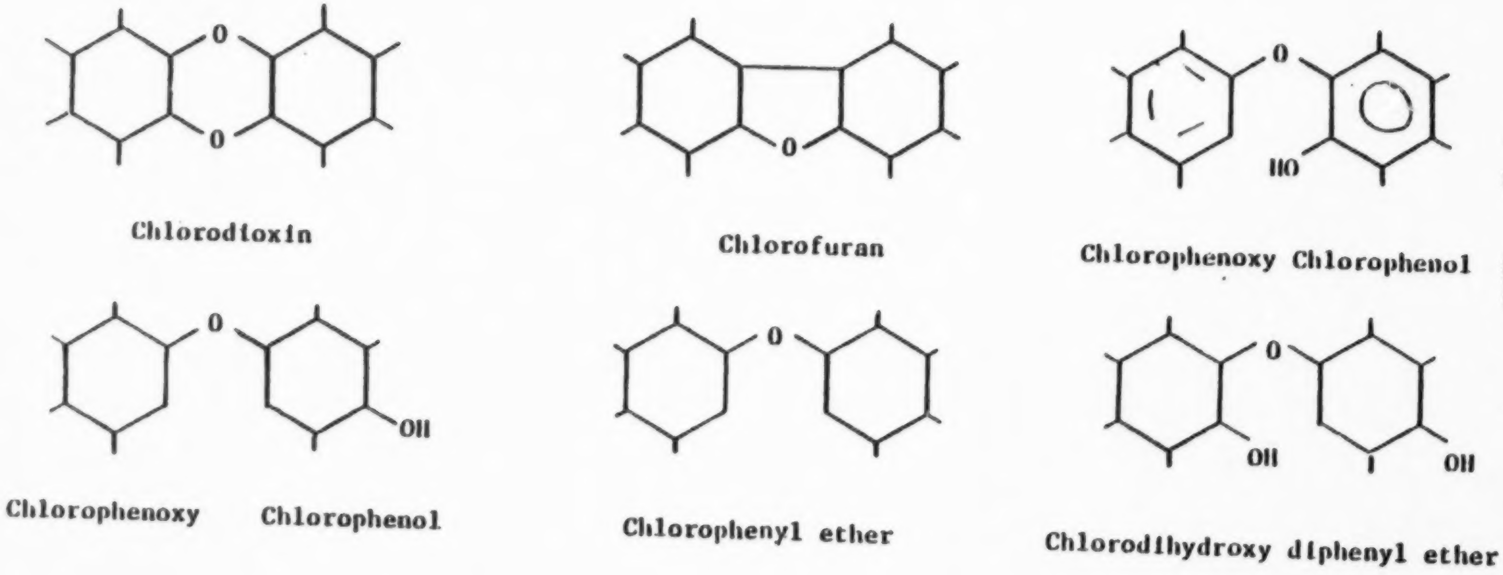
*Most of the information in this appendix is based on the Adhoc Study Group on Pentachlorophenol (1979) and USEPA (1978). Some data in the tables was obtained from USEPA (1978).



Chlorodioxin

Chlorofuran

Chlorophenoxy Chlorophenol

Chlorophenoxy Chlorophenol

Chlorophenyl ether

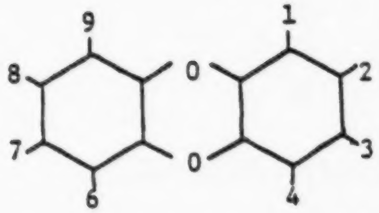
Chlorodihydroxy diphenyl ether

FIGURE A-1. PCP Contaminants - Chlorines at Various Positions on the Rings

Octachlorodibenzodioxin

Heptachlorodibenzodioxins
- 1,2,3,4,6,7,9
- 1,2,3,4,7,8,9

Hexachlorodibenzodioxins
- 1,2,3,7,8,9
- 1,2,3,6,7,8
- 1,2,4,6,7,9
- 1,2,4,6,8,9
- 1,2,3,6,7,9
- 1,2,3,6,8,9



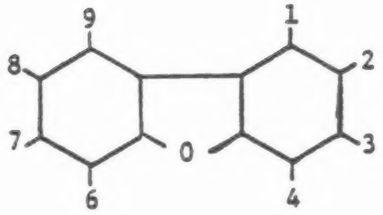
Polychlorodibenzodioxin

TABLE A-1. Dioxin Contaminants Found in PCP heptachlorodibenzofuran

hexachlorodibenzofuran
    1,2,3,6,7,8

pentachlorodibenzofurans
    2,3,4,6,7
    1,2,4,7,8
    2,3,4,7,8

tetrachlorodibenzofurans
    2,3,6,7
    2,4,6,7



Chlorodibenzofuran

TABLE A-2. Dibenzofuran Contaminants in PCP

TABLE A-3. COMPOSITION AND CONCENTRATIONS OF CONTAMINANTS IN PENTACHLOROPHENOL PREPARATIONS

Chemical	PCP Commercial Composite	Monsanto PCP mg/kg	PCP 3-Sample Composite mg/kg	EC-7[1] (mg/kg)	Dowicide 7[2]	PCP[3] (mg/kg)	PCP-Na[2] mg/kg
Pentachlorophenol	85-90%			89.8%	88.4%		
Tetrachlorophenol	5-10%			10.2%	4.4%		
Trichlorophenol	1%			< 1%	.1%		
Chlorinated Phenoxy Phenols	5%				6.2%		
Octachlorodibenzodioxin	~1000 mg/kg	1380	1170	5.5,8.0,15.0	250,2500	160,280,210,370	110,115
Heptachlorodibenzodioxin		520	199	1.1, .6, 6.5	125	125,180,130,130	40
1,2,3,4,6,7,8					171		22
1,2,3,4,6,7,9					63		16
Hexachlorodibenzodioxin		8	11	.15, .03,1.0	4	9.5,9.1,10.0,5.4	3.4
1,2,3,7,8,9					0		.1
1,2,3,6,7,8					5		1.2
1,2,4,6,7,9							
1,2,4,6,8,9							
1,2,3,6,7,9	~100 mg/kg				1		.5
1,2,3,6,8,9	mg/kg				3		1.6
Pentachlorodibenzodioxin							
Tetrachlorodibenzodioxin	No	No	No	No	No	No	No
Octachlorodibenzofuran		260	137	.2,< .1,<1.0	80	105,210,55,65	24
Heptachlorodibenzofuran		400	81	.5,<.1, 1.8	80	95,320,70,60	50
Hexachlorodibenzofuran		90	19	.3,<.03,<1	30	15,36,13,9	11
Pentachlorodibenzofuran		40		.03,<.03		.05,.25,.20,.20	.05
Tetrachlorodibenzofuran				.45,<.02		<.02,.05,.20	<.02

[1] Three analyses.
[2] Two analyses.
[3] Four analyses.

Source: USEPA (1978)

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TABLE A-4. HEXA- AND OCTACHLORODIBENZODIOXIN CONCENTRATIONS IN DOMESTIC PCP'S

		Concentration (mg/kg)	
Sample	Manufacturer	Hexachlorodioxin	Octachlorodioxin
1	Vulcan	10	1700
2	Vulcan	ND	ND
3	Vulcan	15	2500
4	Vulcan	16	3600
5	Reichold	20	700
6	Reichold	17	600
7	Reichold	23	900
8	Reichold	ND	ND
9	Monsanto	15	1400
10	Monsanto	12	1100
11	Monsanto	15	1900
12	Dow	ND	2
13	Dow	ND	2
14	Dow	ND	ND
15	Dow	16	1500
16	Dow	16	1800
17	Dow	21	3400

SOURCE: USEPA (1978)

There is evidence that PCP may be a major source of chlorinated dioxins in the environment. It is probably a lesser source of dibenzofurans. The polychlorinated dibenzodioxin and dibenzofuran contaminants are more stable and persistent, but less mobile in the environment than PCP. This is because PCP has a higher vapor pressure, higher water solubility, and lower adsorption capacity than the other compounds. Even so, these compounds appear capable of degradation in the environment. They are intermediates in PCP degradation and PCP degrades under ideal conditions to innocuous products in at most a couple of months.

There is also evidence that dibenzodioxins and dibenzofurans can migrate from PCP-treated products (wood) into animal tissues and products that are consumed by man. Part of this material may be due to different degradation rates between PCP and other compounds.

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APPENDIX B. BIODEGRADATION PATHWAYS AND PRODUCTS

Many investigators have attempted to define the degradation pathways of pentachlorophenol in soil and to identify the resulting degradation products.

Decomposition products isolated from the soil include 2,3,4,5-, 2,3,5,6-, and 2,3,4,6-tetrachlorophenols; 2,4,5- and 2,3,5-trichlorophenols; 3,4- and 3,5-dichlorophenols; and 3-chlorophenol (Kaufman, 1978). (see Table B-1). Of the three tetrachlorophenol isomers, Kaufman found that 2,3,4,6-tetrachlorophenol degraded most rapidly and concluded that the chlorines ortho and para to the hydroxyl group underwent reductive dechlorination more easily than did the meta chlorine. This finding has been supported by Niki et al. (1971) who detected three tetrachlorophenols in extracts of PCP-treated soil using gas chromotography. Kuwatsuka and Igarishi (1971a, 1971b) also studied degradation of PCP in several soils under various conditions and detected the same tetrachlorophenol compounds, as did Ide et al. (1972), as well as 2,3,6-, 2,4,6-, 2,3,5-, and 2,3,4- and/or 2,4,5-trichlorophenol (see Figure B-1). The appearance of all three isomers of tetrachlorophenol and four or five of six tetrachlorophenol isomers as degradation products of PCP suggests that the degradation in soil is accomplished not only through microbial action but also through chemical reaction, although microbial reaction is the major course.

In addition to these compounds, Kuwatsuka and Igarishi (1975) found pentachloroanisole (PCA) as a PCP degradation product. Cserjesi and Johnson (1972) suggested, however, that although PCA is a metabolic product of PCP, it cannot be considered to be an intermediate in the degradation pathway of PCP because PCA is more resistant to degradation than is PCP. Igarashi et al. (1971, 1978) explained the presence of PCA as a degradation product that was formed and subsequently degraded back to PCP.

Recently, Murthy and Kaufman (1977) have investigated the degradation of PCP to PCP in anaerobic and aerobic soils. In anaerobic soils, 42% of the PCA was reduced back to PCP in 24 days, whereas in aerobic soil, the amount of PCA degradation back to PCP was only 5.6%. These results show that although there is reversion of PCA back to PCP in both types of soils, the reactions involved in the degradation of PCA in anaerobic soil are reductive, thereby leading to a greater percent of PCA returning to PCP. However, in aerobic soils, the reaction is both reductive and oxidative, and a smaller percentage of PCA reverts back to PCP.

The degradation pathway was further defined by studies by Murthy and Kaufman (1977), who showed that PCP was degraded in soil primarily by reductive dehalogenation to tetra-, tri, and dichlorophenols. More detailed studies have shown that the dehalogenation process most often occurs at either the ortho or para position (Kaufman, 1978). In addition to dehalogenation, methylation appears to be a major reaction along the degradation pathway. It has been shown that pentachlorophenol as well

TABLE 8-1. DEGRADATION PRODUCTS OF PENTACHLOROPHENOL

Chemical Name	Source*	Reference
2,5-dichloro-3-hydroxy-6-pentachlorophenoxy-p-benzoquinone	P	Munakata and Kuwahara, 1969
3,4,5-trichloro-6-(2',3',4',5'-tetrachloro-6'-hydroxyphenoxy)-o-benzoquinone	P	Munakata and Kuwahara, 1969
2,5-dichloro-3-hydroxy-6-(2',4',5',6'-tetrachloro-3'-hydroxyphenoxy)-p-benzoquinone	P	Munakata and Kuwahara, 1969
3,5-dichloro-4(2',3',5',6'-tetrachloro-4hydroxy)-6-(3,4,5,6-tetrachloro-2-hydroxyphenoxy)-O-benzoquinone	P	Munakata and Kuwahara, 1969
1,2,3,4,6,7,8,9-octachlorodibenzo-p-dioxin	P	Crosby *et al.*, 1972
2,4,5,6-tetrachloroesorcinol	P	Munakata and Kuwahara, 1969
Chloranilic acid	P	Munakata and Kuwahara, 1969
2,3,4,5-tetrachlorophenol	S	Kuwatsuka and Igarashi, 1975: Ide et al., 1972; Igarashi and Kuwatsuka, 1973; Murthy et al., 1977
2,3,5,6-tatrachlorophenol	S	Kuwatsuka and Igarashi, 1975; Ide *et al.*, 1972; Murthy *et al.*, 1977
2,3,4,6-tatrachlorophenol	S	Kuwatsuka and Igarasni, 1975: Ide *et al.*, 1972
2,4,5-trichlorophenol	S	Kuwatsuka and Igarashi, 1975; Ide *et al.*, 1972
2,3,6-trichlorophenol	S	Kuwatsuka and Igarashi, 1975; Murthy *et al.*, 1977
2,3,4-trichlorophenol	S	Kuwatsuka and Igarashi, 1975
2,3,5-trichlorophenol	S	Kuwatsuka and Igarashi, 1975: Ide *et al.*, 1972; Igarashi and Kuwatsuka, 1973
2,4,6-trichlorophenol	S	Kuwatsuka and Igarashi, 1975
3,4-dichlorophenol	S	Kuwatsuka and Igarashi, 1975; Ide *et al.*, 1972
3,5-dichlorophenol	S	Kuwatsuka and Igarashi, 1975: Ide *et al.*, 1972
2,3,4,5-tetrachloroanisole	S	Ide *et al.*, 1972
2,3,5,6-tetrachloroanisole	S	Ide *et al.*, 1972
2,3,4,6-tetrachloroanisole	S	Ide *et al.*, 1972; Engel *et al.*, 1966
2,3,5-trichloroanisole	S	Ide *et al.*, 1972
2,4,5-trichloroanisole	S	Ide *et al.*, 1972
3,4-dichloroanisole	S	Ide *et al.*, 1972
3,5-dichloroanisole	S	Ide *et al.*, 1972
3-chloroanisole	S	Ide *et al.*, 1972
Pentachloroanisole	S,M	Ide *et al.*, 1972; Kuwatsuka and Igarashi. 1975; Suzuki and Nose, 1971; Cserjesi and Johnson, 1972; Igarashi and Kuwatsuka. 1971; Murthy *et al.*, 1977
tetrachlorocatechol	M	Suzuki, 1977
tetrachlorohydroquinone	M	Suzuki, 1977
tetrachlorohydroquinone	M	Suzuki, 1977
tetrachlorohydroquinone. dimethylether	M	Suzuki and Nose, 1971
tetrachlorobenzoquinone	M	Reiner *et al.*, 1978
2,6-dichlorohydroquinone	M	Reiner *et al.*, 1978
^{14}CO	S,M	Chu and Kirsch, 1972: Kirsch and Etzel. 1973; Suzuki, 1977
Cl-	M	Watanabe, 1973b; Suzuki, 1977
tetrachloromuconic acid		Lyr, 1962
5-hydroxytrichloromuconic acid		Lyr, 1962

*M = isolated microorganisms; P = photodegradation; S - soil.

Source: Kaufman (1978).

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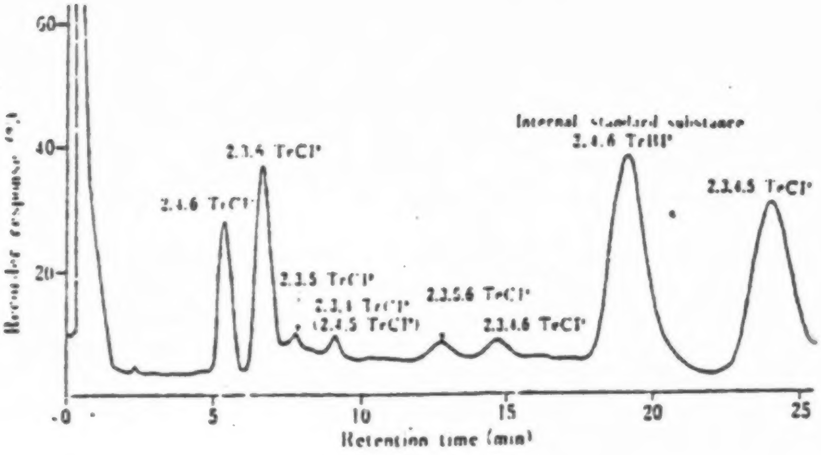
as other chlorophenols formed by dehalogenation are candidates for methylation. Ide *et al*. (1972) found several tetrachloroanisoles and trichloroanisoles as methylated products of PCP in soil. 2,3,4,5-, 2,3,5,6-, and 2,3,4,6-tetrachloroanisole, 3,4-, and 3,5-dichloroanisole, 3-chloroanisole, 2,3,5- and 2,4,5-trichloroanisole; 2,3,6-, 2,5,6-, and 2,4,6-trichloroanisoles were not found. Igarashi and Kuwatsuka (1971), and Matsunaka (1975) propose a degradation pathway of PCP (shown in Figure B-2) that commences with methylation or dechlorination of PCP.

Different degradation products are found when PCP is degraded in soil than when it is degraded by microorganisms isolated from soil . PCP degradation by isolated microorganisms occurs mostly by oxidative mechanisms, whereas degradation in soil is a reductive process. Some oxidation of PCP does occur in soil; Suzuki (1977) reported that $^{14}CO_2$ evolved from PCP-^{14}C-treated soil. Nevertheless, the generalization can be made and is discussed further below.

Suzuki (1977) reported that *Pseudomonas sp*. degraded PCP-^{14}C rapidly and released $^{14}CO_2$ equivalent to 46.8% of the PCP-^{14}C added to the bacterial cell suspension within 1 hour of incubation. Tetrachlorocatechol dimethylether (TCCDM) and tetrachlorohydroquinone dimethylether (TCHQDM) were intermediate metabolites. Tetrachlorocatechol (TCC) and tetrachlorohydroquinone (TCHQ) were also isolated from the incubation medium. Although the production of TCC and TCHQ increased with incubation time, the amount of TCC and TCHQ present at any time was very small. In all cases, the amount of TCC ranged from 0.005% to 0.02% and TCHQ was from 0.2% to 0.5% of the original amount of PCP. Interestingly, when PCP was incubated in a sterilized bacterial suspension, neither TCC nor TCHQ were found, a finding leading to the conclusion that the production of these metabolites was by microbial conversion and not by spontaneous transformation. Suzuki *et al.* (1970) showed that an isolated soil microorganism was a catalyst for the methylation of PCP to PCA and TCHQ to TCHQDM. However, it was not evident that the *Pseudomonas sp*. used by Suzuki (1977) promoted such a methylation of hydroxy groups. Examining soil perfused with 49 mg/l PCP, he found that after an 8-day period of no degradation, chloride ion liberation commenced and continued until the process was completed within 3 weeks. Additional amounts of PCP applied to the soil were degraded without a lag period. A species of *Pseudomonas* isolated from the degradation process was able to use PCP as the sole carbon source, with complete liberation of the chloride ion.

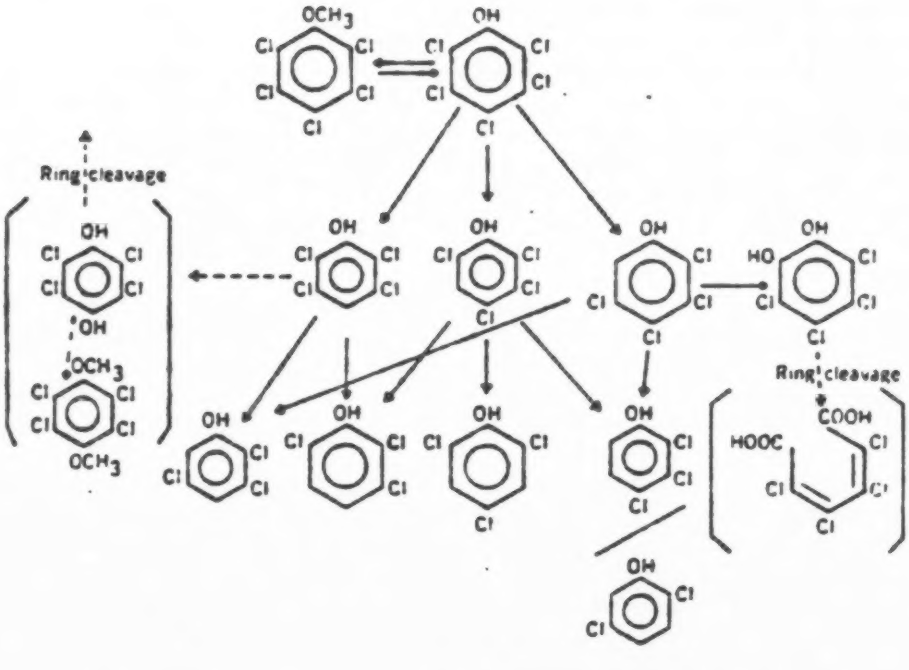
Watanabe (1973) found a strain of *Pseudomonas sp*. to be responsible for the degradation of PCP in soil by a process of dechlorination. Sakomizu, *et al*. (1975) found a similar result of PCP degradation, using the same species *Pseudomonas sp*. isolated from a soil. However, the degradation of PCP by dechlorination was not found by Suzuki (1977), perhaps due to the use of different bacterial species.

Amino acid analysis of the bacterial cells disclosed that the ^{14}C derived from PCP-^{14}C was rapidly incorporated into the cell constituents. The evolution of $^{14}CO_2$ from PCP-^{14}C treated cultures is concrete evidence



Source: Kuwatsuka and Igarishi (1975)

FIGURE B-1 GAS-CHROMATOGRAM OF PCP DEGRADATION PRODUCTS IN ACID FRACTION AFTER 5 DAY'S INCUBATION IN ANJO SOIL


OCH3
OH
Cl
Ring cleavage
OCH3
HO
HOOC
COOH

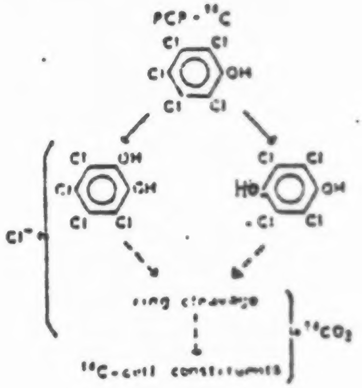
Source: Igarishi and Kuwatsuka (1971)

FIGURE B-2 PROPOSED PATHWAY OF PCP DEGRADATION IN SOIL of the cleavage of the benzene ring (Suzuki, 1977). It is generally accepted that the ring cleavage occurs after the conversion of the substrates to ortho or para dihydroxyphenol derivatives by the metabolism of aromatic compounds by microorganisms. Consequently, it has been suggested that TCC and TCHQ are intermediate metabolites that appear prior to ring cleavage. Both TCC and TCHQ degrade at any stage of the incubation period. A hypothetical pathway of PCP degradation by *Pseudomonas sp.* isolated from a soil is shown in Figure B-3.

Chu *et al.* (1978) agreed with Suzuki's (1977) finding that TCHQ is an intermediate in the degradation of PCP. These authors investigated the metabolism of PCP, using two different bacterial cultures, one parent culture and one mutant culture. For the mutant, PCP was degraded into tetrachlorohydroquinone (TCHQ), tetrachlorobenzoquinone (TCBQ), and tetrachlorohydroxybenzoquinone (TCHBQ). However, kinetic evidence suggests that TCHBQ is not the first quinone to be produced by PCP degradation. Studies of changes to TCHQ under increasing pH and further experimental work led to the conclusion that TCHQ was converted within minutes to TCBQ, which was subsequently transformed to a more stable TCHBQ.

For the parent culture, the rate of this reaction slowed, but did not cease. TCHBQ was subjected to metabolic attack by the parent cultures, but the compound was neither completely dechlorinated nor was the ring ruptured. Chu *et al.* (1978) suggest that another product was formed that was not completely metabolically active. On the other hand, TCBQ was rapidly metabolized by the parent culture and, when applied to the mutant, yielded substantial amounts of 2,6-dichlorohydroquinone (DCHQ). Application of TCHQ to the mutant resulted in the formation of DCHQ, but the transformation of TCHQ to TCHBQ competed strongly with this metabolic activity in either the parent culture or the mutant. These findings confuse data interpretation. PCP degradation apparently involves converting the compound to partially dechlorinated hydroquinone intermediates, which then undergo ring cleavage. The critical intermediates appear to be 2,6-DCHQ, 2,3,5,6-TCHQ, or 2,3,5,6-TCBQ.

Several fungi are capable of degrading PCO. The ability of *Trichoderma* to degrade PCP was first discovered when Duncan and Deverall (1964) found that twice as much PCP was needed to control this organism. They also found that *Trichoderma* was able to degrade PCP at a concentration toxic to a *Basidiomycete*. However, at lower concentrations, wood-staining *Basidiomycetes* were able to degrade PCP. The ability of *Basidiomycetes* to degrade PCP is explained by the presence of phenol oxidases (Lyr, 1963). Wood-rotting *Tramates versicolor* was able to degrade PCP by secreting laccase into the culture medium. Cserjesi (1967), however, identified several *Trichoderma sp.*, which gave negative responses to phenol oxidase tests, but which were able, nonetheless, to degrade PCP. *Penicillium sp.*, *Cephaloascus fragans* and *Graphium sp.* were all capable of degrading PCP, but to a lesser extent than did *Trichoderma sp.* *C. fragans* appeared to have an initial low tolerance to pentachlorophenol, but was able to increase its resistance significantly such that the "adapted" strain was at times able to degrade 11% more of the original concentration of PCP


PCP-^{14}C
Cl
OH
Cl^-
HO
ring cleavage
$^{14}CO_2$
^{14}C-cell constituents

Source: Suzuki (1977)

FIGURE B-3 SUGGESTED METABOLIC FATE OF PCP IN PSEUDOMONAS SP. ISOLATED IN SOIL than did the "wild" strain (Curtis *et al*., 1972) (see Table B-2). PCP degradation has also been attributed to *Aspergillus Sydowi*, *Socpuloriopsis*, and *Penicillium sp*. (Curtis *et al*., 1972).

TABLE B-2. CONCENTRATION OF PENTACHLOROPHENOL IN 2.5% MALT EXTRACT SOLUTION (AS CULTURING MEDIUM) BEFORE AND AFTER 12 DAYS INCUBATION PERIOD WITH VARIOUS FUNGI

Fungus		Concentration (mg/100 ml medium)		
		Original Concentration	Final Concentration	
			"Wild" Strain[1]	"Adapted" Strain[2]
T. harzianum	No. 1	1.05	0.65	0.79
	No. 2	0.77	0.35	0.24
T. viride				
T. viride		1.05	0.05	-
T. virgatum	No. 1	1.05	0.02	-
	No. 2	0.70	0.45	-
Cephaloascus fragrans	No. 1	0.72	0.74	0.72
	No. 2	0.80	0.79	0.70
	No. 3	0.69	0.66	0.66
	No. 4	0.70	0.60	0.68
	No. 5	0.72	0.72	0.73
	No. 6	0.72	0.71	0.74
Graphium sp.	No. 1	0.40	0.38	0.39
	No. 2	0.80	0.76	0.78
Pencillium sp.		0.40	0.38	-
Ceratocystis pilifera		0.40	0.41	-

---

[1] Cultures maintained on malt agar and transferred to the toxic media as required for test.

[2] Cultures grown continuously on malt agar medium with progressively increasing concentration of sodium pentachlorophenate.

Source: Cserjasi (1967).

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